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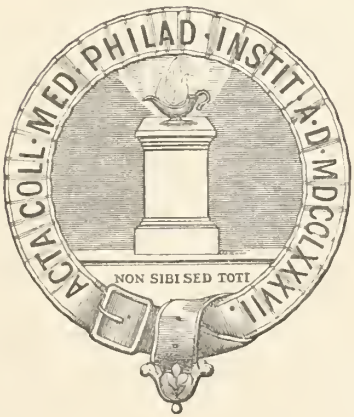

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COLLEGE OF PHYSICIANS OF PHILADELPHIA.

1877.

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11473

LIST

OF THE

PRESIDENTS OF THE COLLEGE FROM THE TIME OF ITS INSTITUTION.

ELECTED

1787. JOHN REDMAN, M.D.
1805. WILLIAM SHIPPEN, M.D.
1809. ADAM KUHN, M.D.
1818. THOMAS PARKE, M.D.
1835. THOMAS C. JAMES, M.D.*
1835. THOMAS T. HEWSON, M.D.
1848. GEORGE B. WOOD, M.D., LL.D.

* Died four months after his election.

FELLOWS

OF THE

COLLEGE OF PHYSICIANS OF PHILADELPHIA.

JULY, 1877.

[Non-Resident Fellows are marked thus (*).]

ELECTED

1870. Adler, John M., M.D.

1859. Agnew, D. Hayes, M.D., LL.D., Professor of Surgery in the University of Pennsylvania, Surgeon to the Pennsylvania Hospital, Consulting Surgeon to the Orthopædic Hospital, and to the Philadelphia and Northern Dispensaries.

1876. Alison, Robert H., M.D., Assistant Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases.

1867. Allen, Harrison, M.D., Professor of Comparative Anatomy and Zoology in the University of Pennsylvania, Surgeon to the Philadelphia Hospital and to St. Joseph's Hospital.

1873. Allis, Oscar H., M.D., Surgeon to the Presbyterian Hospital and to the Howard Hospital.

1869. Andrews, T. Hollingsworth, M.D., Demonstrator of Anatomy in the Jefferson Medical College, Surgeon for Out-Patients to the Pennsylvania Hospital, Surgeon to the Howard Hospital, Consulting Surgeon to the Hospital of the Good Shepherd, Radnor.

1872. Ashbridge, William, M.D., Physician to the German Hospital, Surgeon for Out-patients to the Pennsylvania Hospital.

1863. Ashhurst, John, Jr., M.D., Professor of Clinical Surgery in the University of Pennsylvania, Surgeon to the Episcopal Hospital and to the Children's Hospital, Consulting Surgeon to St. Christopher's Hospital and to the Hospital of the Good Shepherd, Radnor.

ELECTED

1865. Ashhurst, Samuel, M.D.

1835. Ashmead, William, M.D.

1857. Atlee, Walter F., M.D., Consulting Physician and Surgeon to St. Luke's Hospital, Bethlehem.

1846. Atlee, Washington L., M.D.

1852. Bache, Thomas Hewson, M.D.

1876. Baldwin, Louis K., M.D.

1873. Baxter, H. F., M.D.

1874. Beecher, A. C. W., M.D.

1860. Benner, Henry D., M.D.

1874. Bennett, W. H., M.D., Physician to the Episcopal Hospital and to St. Christopher's Hospital.

1871. Bertolet, R. M., M.D.

1851. Biddle, John B., M.D., Professor of Materia Medica and General Therapeutics in the Jefferson Medical College.

*1866. Black, J. J., M.D.

*1867. Boardman, Charles H., M.D.

1859. Boker, Charles S., M.D., Surgeon to St. Joseph's Hospital.

1864. Bolling, Robert, M.D.

1842. Bridges, Robert, M.D., Professor of Chemistry in the Philadelphia College of Pharmacy.

1856. Brinton, John H., M.D., Lecturer on Operative Surgery in the Jefferson Medical College, Surgeon to the Philadelphia Hospital and to St. Joseph's Hospital.

*1851. Bullock, William R., M.D.

1870. Burnett, C. H., M.D., Aurist to the Presbyterian Hospital, Surgeon to the Philadelphia Infirmary for Diseases of the Ear.

1875. Burns, Robert, M.D.

1863. Burpee, David, M.D., Physician to the Howard Hospital.

1868. Cheston, D. Murray, M.D., Physician to the Children's Hospital.

1873. Clark, Leonardo S., M.D., Physician to the Charity Hospital.

ELECTED

1872. Cleemann, Richard A., M.D.

*1842. Clymer, Meredith, M.D.

1827. Coates, Benjamin Hornor, M.D.

1871. Cohen, J. Solis, M.D., Lecturer on Laryngoscopy and Diseases of the Throat and Chest in the Jefferson Medical College, Physician to the German Hospital.

*1870. Corbit, William B., M.D.

*1857. Corse, James M., M.D.

1866. Cruice, R. B., M.D., House Surgeon to St. Joseph's Hospital.

1873. Cruice, W. R., M.D.

1868. Cummiskey, James, M.D.

1858. Da Costa, J. M., M.D., Professor of the Principles and Practice of Medicine in the Jefferson Medical College, Physician to the Pennsylvania Hospital, Consulting Physician to the Children's Hospital and to the Northern Dispensary.

1859. Darrach, James, M.D., Consulting Surgeon to the Germantown Hospital.

1866. Darrach, William, M.D., Physician to the Germantown Hospital.

1874. Deakyne, A. C., M.D.

*1870. Deal, L. J., M.D.

1864. Downs, R. N., M.D., Consulting Physician to the Germantown Hospital.

1864. Duer, E. L., M.D., Accoucheur to the Philadelphia Hospital, Surgeon to the State Hospital for Women and Infants, Visiting Physician to the Preston Retreat.

1871. Duhring, L. A., M.D., Clinical Professor of Skin Diseases in the Hospital of the University of Pennsylvania, Physician to the Dispensary for Skin Diseases.

1863. Dunglison, Richard J., M.D.

*1871. Dunglison, Thomas R., M.D.

*1849. Dunott, Justus, M.D.

1860. Dunton, William R., M.D., Consulting Physician to the Germantown Hospital.

ELECTED

1842. Evans, Charles, M.D.

1868. Evans, H. Y., M.D., Physician to the Charity Hospital.

1872. Finn, W. H., M.D.

1866. Fischer, Emil, M.D.

1862. Forbes, William S., M.D., Surgeon to the Episcopal Hospital.

1870. Ford, W. H., M.D.

1831. Fox, George, M.D.

1864. Fricke, Albert, M.D.

1870. Gardette, E. B., M.D.

1873. Gerhard, George S., M.D., Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases, Assistant Physician to the Children's Hospital, Pathologist to the Presbyterian Hospital.

1864. Getchell, F. H., M.D., Clinical Lecturer on Obstetrics and Diseases of Women in the Jefferson Medical College, Obstetric Physician to the Catharine Street Dispensary.

*1846. Gibbons, Henry, M.D.

1863. Gilbert, W. Kent, M.D.

*1848. Given, Robert A., M.D.

*1854. Gobrecht, William H., M.D.

1868. Goodell, William, M.D., Professor of Clinical Gynæcology in the University of Pennsylvania, Physician in Charge of the Preston Retreat, Consulting Physician to the Lying-in Department of the Northern Dispensary.

1867. Goodman, H. Earnest, M.D., Surgeon to Wills Hospital and to the Orthopædic Hospital, Consulting Surgeon to the State Hospital for Women and Infants.

1864. Granger, William H., M.D.

1857. Green, Alfred, M.D.

1870. Grier, M. J., M.D.

1821. Griffiths, Elijah, M.D.

1842. Griscom, John D., M.D.

ELECTED

1857. Gross, Samuel D., M.D., LL.D., D.C.L. Oxon., Professor of the Principles and Practice of Surgery in the Jefferson Medical College, Consulting Surgeon to the Orthopædic Hospital.

1868. Gross, Samuel W., M.D., Lecturer on Diseases of the Urino-Genital Organs in the Jefferson Medical College, Surgeon to the Philadelphia Hospital, Consulting Surgeon to the Northern Dispensary.

1871. Grove, John H., M.D., Surgeon to St. Mary's Hospital.

1863. Hall, A. Douglass, M.D., Surgeon to Wills Hospital, Ophthalmic Surgeon to St. Mary's Hospital.

1865. Hamilton, George, M.D.

*1859. Hammond, William A., M.D.

1869. Hare, H. B., M.D., Professor of Hygiene in the University of Pennsylvania, Pathological Chemist to the Pennsylvania Hospital.

1865. Harlan, George C., M.D., Surgeon to Wills Hospital, Ophthalmic and Aural Surgeon to the Children's Hospital.

1863. Harlow, Lewis D., M.D.

1862. Harris, Robert P., M.D., Physician to the Franklin Reformatory Home.

1847. Hartshorne, Edward, M.D.

*1851. Hartshorne, Henry, M.D.

1868. Hassler, Ferdinand A., M.D.

*1849. Hastings, John, M.D.

1855. Hatfield, Nathan L., M.D., Consulting Physician to the Northern Dispensary.

*1865. Hayes, Isaac I., M.D.

1835. Hays, Isaac, M.D.

1872. Hays, I. Minis, M.D.

1853. Hewson, Addinell, M.D.

1872. Hinkle, A. G. B., M.D.

ELECTED

1863. Hodge, H. Lenox, M.D., Demonstrator of Anatomy in the University of Pennsylvania, Surgeon to the Presbyterian Hospital and to the Children's Hospital, Consulting Physician to the Lying-in Department of the Northern Dispensary, Consulting Surgeon to the Hospital for Relief of Diseases of the Rectum and Urinary Organs.

1852. Hooper, William H., M.D.

1867. Horn, George H., M.D.

1868. Howell, Samuel B., M.D., Professor of Mineralogy and Geology in the University of Pennsylvania.

1854. Hunt, William, M.D., Surgeon to the Pennsylvania Hospital and to the Orthopædic Hospital.

1871. Hunter, Charles T., M.D., Demonstrator of Surgery in the University of Pennsylvania, Surgeon for Out-patients to the Pennsylvania Hospital.

1863. Hutchinson, James H., M.D., Physician to the Pennsylvania Hospital and to the Children's Hospital.

1871. Ingham, James V., M.D., Obstetrician to the State Hospital for Women and Infants.

1871. Jenks, William F., M.D., Surgeon to the State Hospital for Women and Infants, Consulting Physician to the Lying-in Department of the Northern Dispensary.

1864. Jones, S. P., M.D., Assistant Physician to the Pennsylvania Hospital for the Insane.

1867. Judson, Oliver A., M.D.

1849. Keating, William V., M.D., Physician to St. Joseph's Hospital.

1867. Keen, William W., M.D., Surgeon to St. Mary's Hospital.

1852. Kennedy, Alfred L., M.D.

*1844. King, Charles R., M.D.

1864. King, William M., M.D., U. S. N.

1875. Kirkbride, J. J., M.D., Physician for Out-patients to the Pennsylvania Hospital.

1839. Kirkbride, Thomas S., M.D., Physician-in-Chief to the Pennsylvania Hospital for the Insane.

1848. Klapp, Joseph, M.D., Physician to the Howard Hospital.

ELECTED

*1865. LaRoche, C. Percy, M.D.

1868. Leavitt, Thaddeus L., M.D., Physician to the Germantown Hospital.

1864. LeConte, John L., M.D.

1864. Leedom, John M., M.D.

1851. Leidy, Joseph, M.D., LL.D., Professor of Anatomy in the University of Pennsylvania.

1877. Lewis, Morris J., M.D., Assistant Physician to the Children's Hospital, and to the Orthopædic Hospital and Infirmary for Nervous Diseases.

1855. Lewis, Francis W., M.D.

1849. Lewis, Samuel, M.D.

1836. Littell, S., M.D., Consulting Surgeon to the Philadelphia Dispensary, Emeritus Surgeon to Wills Hospital.

*1847. Logan, J. Dickinson, M.D.

1849. Ludlow, John L., M.D., Physician to the Philadelphia Hospital and to the Presbyterian Hospital.

1875. McClellan, George, M.D.

1871. McFerran, J. A., M.D., Physician to the Gynæcological Hospital and Infirmary for Diseases of Children.

1866. Maury, F. F., M.D., Lecturer on Venereal and Cutaneous Diseases in the Jefferson Medical College, Surgeon to the Philadelphia Hospital, Consulting Surgeon to the State Hospital for Women and Infants.

*1850. Mayer, Edward H., M.D.

1868. Mears, J. Ewing, M.D., Demonstrator of Surgery in the Jefferson Medical College, Surgeon to St. Mary's Hospital.

1875. Meigs, Arthur V., M.D., Assistant Physician to the Children's Hospital, Examining Physician to the Hospital of the Good Shepherd, Radnor.

1856. Meigs, James Aitken, M.D., Professor of the Institutes of Medicine and of Medical Jurisprudence in the Jefferson Medical College, Physician to the Pennsylvania Hospital.

1843. Meigs, John Forsyth, M.D., Physician to the Pennsylvania Hospital, Consulting Physician to the Children's Hospital.

ELECTED

1856. Mitchell, S. Weir, M.D., Physician to the Orthopædic Hospital and Infirmary for Nervous Diseases, Consulting Physician to the State Hospital for Women and Infants.

1842. Moehring, Gotthilf, M.D.

1863. Morehouse, George R., M.D., Physician to St. Joseph's Hospital.

1839. Morris, Caspar, M.D.

1856. Morris, J. Cheston, M.D.

1861. Morton, Thomas G., M.D., Surgeon to the Pennsylvania Hospital, to the Orthopædic Hospital, and to the Jewish Hospital, Emeritus Surgeon to Wills Hospital.

1864. Moss, William, M.D.

1865. Nebinger, Andrew, M.D., Physician to St. Mary's Hospital.

1846. Neill, John, M.D.

1869. Norris, Herbert, M.D., Physician to the Catharine Street Dispensary.

1865. Norris, Isaac, Jr., M.D.

1870. Norris, John C., M.D.

1866. Norris, William F., M.D., Clinical Professor of Diseases of the Eye in the Hospital of the University of Pennsylvania, Surgeon to Wills Hospital.

1858. Packard, John H., M.D., Surgeon to the Episcopal Hospital.

1863. Page, Edward A., M.D., Surgeon to St. Joseph's Hospital.

1835. Pancoast, Joseph, M.D., Emeritus Professor of General, Descriptive, and Surgical Anatomy in the Jefferson Medical College.

1864. Pancoast, William H., M.D., Professor of General, Descriptive, and Surgical Anatomy in the Jefferson Medical College, Surgeon to the Philadelphia Hospital, Consulting Surgeon to the Charity Hospital.

*1854. Parrish, Joseph, M.D.

*1835. Paul, John Marshall, M.D.

ELECTED

1836. Paul, John Rodman, M.D.

1839. Peace, Edward, M.D.

1854. Penrose, R. A. F., M.D., LL.D., Professor of Obstetrics and Diseases of Women and Children in the University of Pennsylvania, Consulting Obstetrician to the State Hospital for Women and Infants, Visiting Physician to the Preston Retreat.

1868. Pepper, William, M.D., Professor of Clinical Medicine in the University of Pennsylvania, Physician to the Philadelphia Hospital and to the Children's Hospital.

1872. Porter, William G., M.D., Surgeon to the Presbyterian Hospital and to the Philadelphia Hospital.

1868. Ray, Isaac, M.D.

1866. Reed, Thomas B., M.D., Surgeon to the Presbyterian Hospital, Microscopist to the Philadelphia Hospital.

1849. Reed, Thomas S., M.D.

1842. Reese, John J., M.D., Professor of Medical Jurisprudence in the University of Pennsylvania, Physician to St. Joseph's Hospital, and to the Gynæcological Hospital and Infirmary for Diseases of Children.

1853. Rhoads, James E., M.D.

1871. Richardson, Elliott, M.D., Lecturer on Practical Obstetrics in the University of Pennsylvania, Surgeon for Out-patients to the Pennsylvania Hospital, Obstetric Physician to the Philadelphia Dispensary, Physician to the Catharine Street Dispensary.

1869. Richardson, Joseph G., M.D., Demonstrator of Histology in the University of Pennsylvania, Physician to the Presbyterian Hospital, Physican for Out-patients and Microscopist to the Pennsylvania Hospital.

*1857. Richardson, Thomas G., M.D.

1867. Roberts, Jacob, M.D.

1843. Rodman, Lewis, M.D., Visiting Physician to the Preston Retreat.

1857. Rogers, Robert E., M.D., Professor of Chemistry in the Jefferson Medical College.

1838. Ruschenberger, W. S. W., M.D., U. S. N.

ELECTED

*1852. Sargent, Fitz William, M.D.

1864. Sargent, Winthrop, M.D.

1866. Schaffer, Charles, M.D.

1870. Schell, Henry S., M.D., Surgeon to Wills Hospital, Ophthalmic and Aural Surgeon to St. Mary's Hospital.

1875. Seyfert, Theodore H., M.D., Physician to the Gynæcological Hospital and Infirmary for Diseases of Children.

1877. Shakespeare, Edward O., M.D., Lecturer on Refraction and Accommodation of the Eye, and on Operative Ophthalmic Surgery, in the University of Pennsylvania.

1868. Shapleigh, E. B., M.D.

1876. Shippen, Edward, M.D., U. S. N.

1870. Silliman, Henry R., M.D.

1873. Simpson, James, M.D.

1872. Sinkler, Wharton, M.D., Physician to the Episcopal Hospital, and to the Orthopædic Hospital and Infirmary for Nervous Diseases.

1857. Slocum, Alfred M., M.D.

1863. Smith, Albert H., M.D., Physician to and Lecturer on Obstetrics in the Philadelphia Lying-in Charity, Visiting Physician to the Preston Retreat, Consulting Physician to the Hospital of the Good Shepherd, Radnor.

1863. Smith, A. K., M.D., U. S. A.

1864. Smith, Edward A., M.D.

1845. Smith, F. G., Jr., M.D., Emeritus Professor of the Institutes of Medicine in the University of Pennsylvania.

*1856. Smith, R. K., M.D.

1870. Smyth, Francis G., M.D.

1864. Spooner, Edward A., M.D.

1875. Starr, Louis, M.D., Physician to the Episcopal Hospital and Assistant Physician to the Children's Hospital.

1835. Stewardson, Thomas, M.D.

1842. Stillé, Alfred, M.D., LL.D., Professor of the Theory and Practice of Medicine in the University of Pennsylvania, Consulting Physician to the State Hospital for Women and Infants.

ELECTED

1846. Stocker, Anthony E., M.D.

1871. Strawbridge, George, M.D., Clinical Professor of Diseases of the Ear in the Hospital of the University of Pennsylvania, Surgeon to Wills Hospital, Ophthalmic Surgeon to the Presbyterian Hospital, Surgeon to the Eye and Ear Department of the Philadelphia Dispensary.

1855. Stroud, William D., M.D.

1867. Taylor, R. R., M.D.

1867. Thomas, Charles H., M.D.

1873. Thomas, Richard, M.D.

1869. Thomson, William, M.D., Lecturer on Ophthalmology in the Jefferson Medical College.

1852. Tiedemann, Henry, M.D.

*1853. Tilden, W. P., M.D.

1870. Townsend, Ralph M., M.D.

1850. Townsend, Richard H., M.D.

1870. Turner, A. Paul, M.D.

1866. Tyson, James, M.D., Professor of General Pathology and Morbid Anatomy in the University of Pennsylvania, Physician to the Philadelphia Hospital.

*1852. Tyson, James L., M.D.

1864. Vandyke, E. B., M.D.

1873. Van Harlingen, Arthur, M.D., Assistant Physician to the Dispensary for Skin Diseases.

1852. Wallace, Ellerslie, M.D., Professor of Obstetrics and Diseases of Women and Children in the Jefferson Medical College, Consulting Obstetrician to the State Hospital for Women and Infants, Visiting Physician to the Preston Retreat, Consulting Physician to the Lying-in Department of the Northern Dispensary.

1873. Wallace, W. H., M.D.

*1839. Warrington, Joseph, M.D.

1875. Webb, William H., M.D.

ELECTED

1863. Wells, W. Lehman, M.D.

1864. West, Hilborne, M.D.

1868. Williams, Horace, M.D., Obstetrician to the State Hospital for Women and Infants, Obstetric Physician to the Philadelphia Dispensary, Physician to the Howard Hospital.

1851. Wilson, Ellwood, M.D., Consulting Physician to the Philadelphia Lying-in Charity, Visiting Physician to the Preston Retreat.

1874. Wilson, J. C., M.D., Physician to the Philadelphia Hospital.

1871. Wistar, Thomas, M.D.

1848. Wister, Caspar, M.D., Consulting Physician to the Philadelphia Dispensary.

1852. Wister, Owen Jones, M.D.

1827. Wood, George B., M.D., LL.D., Emeritus Professor of the Theory and Practice of Medicine in the University of Pennsylvania.

1865. Wood, Horatio C., Jr., M.D., Professor of Materia Medica and Pharmacy in the University of Pennsylvania, and Clinical Professor of Diseases of the Nervous System in the Hospital of the same, Physician to the Philadelphia Hospital.

1866. Woods, D. F., M.D., Physician to the Presbyterian Hospital.

1860. Wurts, Charles Stewart, M.D.

1861. Yarrow, Thomas J., M.D., Physician to St. Mary's Hospital.

*1840. Zantzinger, William S., M.D.

[It is particularly requested that any change of appointment, etc., may be communicated to the Committee of Publication before the first of July, in each year, in order that the List may be made as correct as possible.]

ASSOCIATES.

[Limited to Fifty, of whom Twenty may be Foreigners.]

ELECTED

1873. Acland, Henry W., M D., F.R.S., Oxford, England.

1869. Alvarenga, P. F. Da Costa, Lisbon, Portugal.

1847. Atlee, John L., M.D., Lancaster, Pennsylvania.

1876. Baldwin, W. O., M.D., Montgomery, Alabama.

1877. Barnes, Robert, M.D., London, England.

1876. Barker, Fordyce, M.D., New York.

1876. Bigelow, Henry J., M.D , Boston, Massachusetts.

1821. Bigelow, Jacob, M.D., Boston, Massachusetts.

1876. Billings, John S., M.D., Washington, District of Columbia.

1876. Bowditch, Henry I., M.D., Boston, Massachusetts.

1865. Butcher, R. G. H., M.D., Dublin, Ireland.

1877. Byford, William H., M.D., Chicago, Illinois.

1877. Chaillé, Stanford E., M.D., New Orleans, Louisiana.

1848. Christison, Sir Robert, Bart., M.D., D.C.L., LL.D., Edinburgh, Scotland.

1853. Churchill, Fleetwood, M.D., Dublin, Ireland.

1876. Clark, Alonzo, M.D., New York.

1876. Comegys, C. G., M.D., Cincinnati, Ohio.

1876. Corson, Hiram, M.D., Norristown, Pennsylvania.

1876. Davis, N. S., M.D., Chicago, Illinois.

ELECTED

1876. Donaldson, F., M.D., Baltimore, Maryland.
1868. Flint, Austin, M.D., New York.
1857. Gintrac, E., Bordeaux, France.
1876. Green, Traill, M.D., Easton, Pennsylvania.
1852. Hall, Archibald, M.D., Canada.
1868. Hamilton, Frank H., M.D., LL.D., New York.
1876. Hodgen, John T., M.D., St. Louis, Missouri.
1876. Jackson, J. B. S., M.D., Boston, Massachusetts.
1874. Jackson, J. Hughlings, M.D., London, England.
1876. Johnson, George, M.D., F.R.S., London, England.
1876. Johnston, Christopher, M.D., Baltimore, Maryland.
1876. Jones, Joseph, M.D., New Orleans, Louisiana.
1876. Kinloch, R. A., M.D., Charleston, South Carolina.
1876. King, James, M.D., Pittsburgh, Pennsylvania.
1854. Lever, John M., M.D., London, England.
1877. Lister, Joseph, F.R.S., Edinburgh, Scotland.
1865. Macleod, G. H. B., M.D., Edinburgh, Scotland.
1876. Moore, E. M., M.D., Rochester, New York.
1876. Mowry, R. B., M.D., Alleghany City, Pennsylvania.
1873. Ogle, John W., M.D., London, England.
1874. Paget, Sir James, Bart., D.C.L., LL.D., F.R.S., London, England.
1842. Parker, Peter, M.D., China.
1876. Parker, Willard, M.D., New York.
1873. Peacock, Thomas B., M.D., London, England.
1876. Pollock, A. M., M.D., Pittsburgh, Pennsylvania.
1876. Porcher, F. Peyre, M.D., Charleston, South Carolina.
1869. Valcourt, Th. de, Cannes, France.
1857. Valery, Gaetano, Florence, Italy.
1861. Velasco, Pedro Gonzales, Madrid, Spain.
1860. Walther, Hermann, Dresden, Saxony.

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NOTICE.

The present volume of Transactions contains the papers read before the College from October, 1876, to July, 1877, inclusive.

The Committee of Publication thinks it proper to say that the College holds itself in no way responsible for the statements, reasonings, or opinions set forth in the various papers published in its Transactions.

CASE

OF

THROMBOSIS OF THE CEREBRAL VEINS.

By

ARTHUR V. MEIGS, M.D.,

ASSISTANT PHYSICIAN TO THE CHILDREN'S HOSPITAL, EXAMINING PHYSICIAN TO THE HOSPITAL OF THE GOOD SHEPHERD, RADNOR.

[Read October 4, 1876.]

I WISH to present to the Fellows of the College, this evening, the history of a case of thrombosis of the cerebral veins. The patient was under the charge of my father, Dr. J. Forsyth Meigs, in the Pennsylvania Hospital.

M. B., aged 20 years, was admitted into the hospital Oct. 12, 1875. She was born in Ireland, is single, and her occupation is that of servant in a boarding house. She was brought into the house in a semi-comatose condition, so no history can be obtained from her personally. Her friends say that she has only been in America for a few months, and that she was "brought out here," that is, that her passage was paid for, by a man to whom she was engaged to be married. She has been working very hard to pay back the money, but soon after her arrival in this country, her lover began to neglect her. Between the mental depression caused by her lover's desertion, and the hard work she has done, she has been declining in health since about August.

Five days before admission into the hospital she was

seized with vomiting and headache. The headache continued for three days, when it ceased and she seemed better. The day before she was brought into the hospital she became very drowsy and stupid. Her bowels are said to have been regular, and she has had no difficulty in urination. Her menses have been regular also.

On admission, the decubitus is right lateral, the face is drawn into a frown, and the complexion is very yellowish. When loudly spoken to she partially wakes up, and mumbles unintelligibly, but at once relapses into stupor. The head is a shade retracted, and the back of the neck is stiff. She cannot protrude the tongue, and only partially opens the mouth. She can move both arms when disturbed, but the right is generally profoundly still, while the left is kept in constant motion when anything irritates her. The left pupil is slightly more dilated than the right, and the right side of the face is partially paralyzed, not being so much moved as the left. She gets out of bed automatically to urinate. The abdomen is rather flat and yellow. There is somewhat less resonance on percussion under the left than under the right clavicle. The temperature in the evening was 100°. She was ordered a cod-liver oil mixture (25 per cent.), a tablespoonful, with two and a half grains of iodide of potassium, every three hours.

Oct. 13. Temperature, A. M. 99°; P. M. 98°.

Oct. 14. Lies constantly on the right side. Protrudes the tongue to-day when asked to; it is coated, but moist, and the teeth are covered with sordes. The face is slightly drawn to the left. There is no ptosis, and she moves the eyeballs perfectly. She is not so stupid, having asked for some tea. Temperature, A. M. 99°; P. M. 99°.

Oct. 15. The urine is pale and turbid, is acid, and has a sp. gr. of 1025. It contains neither albumen nor sugar. She is delirious, and the saliva runs from her mouth during sleep. Bowels have not been opened since her admission. Temperature, A. M. 99°; P. M. 99°.

Oct. 16. The head is decidedly retracted. Examination

of the heart reveals nothing. The left arm is in a constant state of jactitation, while the right is comparatively quiescent. She still gets up to urinate, but the bowels have not been opened. Temperature, A. M. 99°; P. M. 98°.

Oct. 17. At the morning visit she is found sitting up in bed, and she says that her head is sore. Aided by an injection, she has had a small hard stool. Her whisky is ordered to be increased from three to four fluidounces. Ordered compound gentian mixture, a dessertspoonful (containing 1¾ grains of ammonio-citrate of iron), every two hours. She takes three fluidounces of milk with one of lime-water every two hours. Temperature, A. M. 98°; P. M. 98°.

Oct. 18. She can follow any object moved before the eyes perfectly. Corrugates the left eyelid more than the right, when told to shut the eyes tightly. Moves the legs about equally if the feet are tickled. She seems more conscious, and still gets up. She was given ten grains of bromide of potassium last night, and in future is to have ten grains of chloral with twenty of bromide, every night. Temperature, A. M. 99°; P. M. 98°.

Oct. 20. The movements are very choreic. Is much more intelligent and can understand what is said to her, although unable to make all the motions she wishes. The percussion note beneath the two clavicles seems about equal. Temperature, A. M. 97°; P. M. 98.5°.

Oct. 21. Her eyes are examined ophthalmoscopically by Dr. J. A. Lippincott, and he thinks there is optic neuritis, but the examination is very difficult on account of the restlessness of the patient.

Oct. 22. She seems much better, is perfectly intelligent, and talks without any difficulty. She was given an enema and had a hard passage after it.

Oct. 23. She was found this morning completely comatose, and the choreic movements have ceased. There is opisthotonos, with occasional clonic spasms, during which she breathes stertorously. The spasms seem to be brought on by any irritation. The eyes are staring downwards, and the lids are

partially closed. There is profuse sweating, and incontinence of urine; the pulse is frequent, and the conjunctivæ are insensible. She complained of headache yesterday evening, and the bad symptoms came on during the night. She died at 5 P. M.

The post-mortem examination was made by Dr. Longstreth, the curator, 4½ hours after death. Rigor mortis well established. There was no effusion into either pleural sac. The left lung was adherent, and congested posteriorly, as was the right in a slight degree. The tissue of both lungs pale and anæmic. The pericardium contained a small amount of clear serum, but was smooth and not inflamed. The vena cava and the right auricle and ventricle filled with soft black clots. The right ventricle was not, but the left heart was, firmly contracted. The walls and valves normal. The liver was normal. The spleen rather small and pale. The kidneys presented no abnormal appearance. The uterus retroverted, and some mucus escaping from the cervix.

The skullcap was rather thin. The dura mater not congested, but adherent posteriorly in the median line. The Pacchionian bodies not marked. The dura mater easily reflected, there being no marked congestion except along the longitudinal sinus. The surface of the hemispheres rather dry and sticky. The convolutions decidedly flattened, and pressed closely together, most conspicuously so in front and superiorly, although markedly so everywhere. The arachnoid streaked with white opacities over the posterior perforated space, and between the lobes of the cerebellum. The vessels of the circle of Willis apparently normal. The sinuses of the dura mater contained some blood, but no clots could be found in them. On opening the right lateral ventricle, abundant blood-stained serum escaped. The septum lucidum was destroyed. The right choroid plexus showed its veins filled with firm black clots, although the general surface did not appear congested. There was a firm black clot in the vein coming from the right corpus striatum, and

the venæ Galeni were plugged. The clot continued as far as the straight sinus. The left choroid plexus had one or two veins filled with clot, and these were towards its continuation into the posterior and middle cornua. The color of the villous processes of the membrane was light pink. The body of the fornix was so softened that it broke in the attempt to reflect it. The right corpus striatum appeared of a dusky red color, maintained its outline, but was almost diffluent, easily breaking down under the finger. On section, the dusky-red color was found about half an inch in depth; below, the tissue, although much softened, was firmer and of natural hue. The change of color between the two areas was very sudden. The right optic thalamus was completely disorganized, and in the softened tissue there were minute blood clots. The walls of other parts of this lateral ventricle were markedly softened, but less so than the parts described. In the left lateral ventricle the softening of the walls was very general, especially marked at the corpus striatum and the roof of the posterior and middle cornua.

It was difficult to make out the boundaries of the third ventricle, owing to the destruction of the neighboring parts. When examining the base of the brain, the floor of the third ventricle ruptured spontaneously, and a considerable quantity of fluid escaped. A large area of softening was found in the middle and posterior lobes of the left hemisphere, extending from the surface to the posterior and middle cornua of the lateral ventricle. The softening was most marked superficially, and extended from the level of the ventricle towards the base. Portions of this area were diffluent, and after section slowly drained away, leaving islets of firm white substance. The veins running between the convolutions of this area were found filled with black clots. The cerebellum was normal.

Dr. Wm. Hunt, who saw the brain, notes as follows: Above the corpus callosum, nothing marked beyond injection; below, on the right, very extensive softening all along the borders of

the ventricles, which were almost obliterated by the broken-down material in them. The corpus striatum and the thalamus were almost entirely broken down. On the left, the softening was confined to the middle and posterior lobes. There was thrombus on the right border of the velum interpositum. The choroid plexus was enormously injected, and showed large bead-like deposits in its walls, almost completely filling the middle and posterior horns of the lateral ventricle on the left.

Dr. W. F. Norris examined the eyes, and has kindly given me the following memorandum of the result of his examination. "The posterior halves of the eyes, and the optic nerves in their entire length, were removed. The latter presented at their distal extremities slight pyriform dilatations of their outer sheaths. The optic discs were prominent and very hazy; their normal outlines completely veiled. The retinal veins, dilated with blood, were seen emerging from the centre of the disc, but were completely hidden by the swollen and opaque nerve-fibres as they traversed it, and made their appearance once more as they left it to spread out in the retina. The specimen was hardened in Müller's fluid, and subsequently sections were made of the nerve disc, which were stained with carmine and mounted in balsam. They show that the intraocular end of the nerve was swollen in every diameter and permeated by numerous bloodvessels. The arteries and capillaries were mostly empty, and the veins so gorged with blood as to form a natural injection of the part. On the dried and mounted specimen the distance from the choroid to the summit of the swollen disc measured one-thirty-fourth of an inch."

This case is interesting because the diagnosis was a very difficult one, and because the disease presented itself under a rare form, and apparently had an unusual cause.

There is an article in the *Journal Hebdomadaire*,

for 1829, entitled "*Maladies des Sinus de la Dure-mère*," by Tonnelé, in which he describes thrombosis of the sinuses caused by phlebitis, and simple thrombosis from depressing causes, such as rachitis and malarial diseases; a third form caused by mechanical obstruction, and lastly a fourth, caused by suppurative disease of any part in close connection with the intracranial bloodvessels. Bouchut (*Journal für Kinderkrankheiten*, 1868) mentions two cases of thrombosis in children, in one of which he says the diagnosis was arrived at by means of an ophthalmoscopic examination of the eyes. The most exhaustive article on the subject that I have been able to find, is one by Dr. John A. Lidell on "Thrombosis of the Cerebral Veins and Sinuses of the Dura Mater," in the January and July numbers of the *American Journal of the Medical Sciences*, for 1874. He gives 128 cases, some observed by himself and others collected from various works, and mentions the fact, which I have been unable to find noticed in any other work, that thrombosis of the cerebral veins may occur while yet the sinuses remain free. He says (in speaking of a case) "but . . . the longitudinal and other sinuses of the dura mater did not contain any coagula; and thus it is conclusively shown that thrombosis of the cerebral veins attended with very striking morbid changes and consequences, may occur without there being any thrombosis of the corresponding sinuses of the dura mater. . . . The reader's attention is especially called to this point, because it is a new one, and possesses considerable interest." I do not think that in any of Dr. Lidell's cases the thrombosis and softening were so

extensive as in that which I have presented to the College.

With regard to the cause of the coagulation, it really seems as if it had been produced by the mental distress undergone. The history of the patient reveals none of the usual causes; there was no suppuration of any part, nor any chronic exhausting disease. On the contrary, she had always been a healthy woman. And why should not constant mental depression and anxiety gradually produce general weakness and debility, and thinning of the blood, until finally it might coagulate in the veins?

The diagnosis was a very difficult one to make. No more so, I suppose, than in the case of many other brain lesions, but for the fact that the disease being a very rare one we cannot know it as well as other diseases which are more common. Dr. Meigs was able, a day or two before the death of the patient, to pretty well satisfy himself that the case was one of cerebral thrombosis. At first it presented many of the symptoms of tubercular meningitis; severe headache, and occasional unprovoked vomiting, following failure of general health for some months. The patient was young; her temperature was only very moderately elevated; and her circulation was quiet. All this was very like effusion following tubercular meningitis. But the paralysis was unlike that usual in tubercular disease; it was hemiplegic, and the hemiplegia was quite developed although incomplete.

After a few days the conditions seemed even less like those of meningitis. The patient became quite intelligent; was able to answer questions, although slowly; and seemed to understand perfectly what was

said to her. Then there was the irregularity of the voluntary movements. The muscles of the left side of the body jerked so much as to cause the case to resemble a high degree of chorea. This condition was very striking, and taken in connection with the return of intelligence and the power to sit up in, and even get out of, bed, seemed to demand some other explanation than that of tubercular meningitis. In seeking such other explanation, it was recollected that the patient was said to have been a blooming, healthy-looking girl when she landed in America. Soon after her arrival she was exposed to bitter disappointment and mortification, being deserted by a young man to whom she was engaged to be married. From that time she began to decline in health, losing appetite, strength and color, and soon becoming very anæmic. The idea suggested itself that perhaps the thin blood, and slow and languid circulation, might have allowed the deposition of fibrin and consequent formation of thrombi in some of the cerebral vessels. Then would follow deficient blood-supply to certain areas of the brain, anæmia of those parts, and finally softening.

The termination of the case was by what the French call *apoplexie foudroyante*, effusion into the ventricles of the brain. In a single night the patient became perfectly comatose, the choreic movements ceased, and there was opisthotonos with occasional clonic spasms, the eyeballs being strongly drawn downwards. Of course death took place in a few hours.

I have been unable to find any published account of a case precisely resembling the one I have detailed. It is very striking on account of the absolute freedom of the patient from any previous disease, the very

great amount of clot in the veins while there was none in the sinuses, and the extensive softening. Dr. Lidell gives only two cases in which there were no thrombi in the sinuses, and in neither of them were the clots or softening very extensive. In both instances the patients had suffered from previous disease, one having had an injury of the arm which had caused extensive suppuration, and the other having had cirrhosis and ascites.

CASE

OF

LACERATED WOUND OF THE ELBOW-JOINT,

TREATED SUCCESSFULLY BY THE ANTISEPTIC METHOD OF PROFESSOR LISTER.

By

J. EWING MEARS, M.D.,

SURGEON TO ST. MARY'S HOSPITAL, ETC.

[Read October 4, 1876.]

So far as may be judged from the publication of reports of cases, both public and private, it may be safely asserted that the antiseptic method of treating wounds, either simple or complicated, has not been largely employed in this country. Whether the failure to carry this plan into practice has arisen from any preconceived prejudices, or from a want of appreciation of what may be esteemed its merits, it is difficult to say. It can scarcely be attributed to difficulties presented in obtaining the proper appliances, or in securing their proper application. There appears to be no evidence on record showing that, at the time of its introduction into our public and private practice, the failures to accomplish successful results were so numerous as to create a prejudice against its use. It occurs to me to suggest that, in many cases, too much had been expected of this plan; without doubt,

it was often applied improperly, and under such conditions as necessarily involved failure. In the early accounts given of this method, its distinguished author distinctly stated that a perfect result could not always be expected. More or less suppuration may occur in wounds under the antiseptic application, and should not deter the surgeon from persevering in his treatment. In the management of the case which I bring before the College on this occasion, I followed the instructions given above, and to this fact is due very largely, if not entirely, the successful result obtained.

The patient, M. H., æt. 22, was admitted into the Surgical Wards of St. Mary's Hospital on July 6, with a punctured and lacerated wound of the left elbow-joint, caused by being stepped upon by a mule, the long, pointed heels of the shoe penetrating the cavity of the joint. On examination, some hours after the receipt of the injury, there were found two wounds, one on the outer aspect of the arm, just above the condyle, extending in depth to the bone, but not communicating with the articulation, and the other, on the anterior surface of the joint, quite near to the inner border. This wound opened directly into the joint, the ligament being ruptured to a large extent, and thus exposing the articular surfaces of the bones which were partially denuded of cartilage.

Although the patient had been run over at the time of the receipt of the injury to the joint, the wheels of an empty ice-wagon passing over his chest and right arm, still he was remarkably free from any symptoms of shock. After consultation with my colleagues, it was determined that an effort should be made to save the limb, and, in order to accomplish this end, I decided to employ the antiseptic method. Delay occurred in procuring a portion of the dressing I wished to apply, and the treatment was therefore not begun

until nearly forty-eight hours after the accident, when some inflammatory action had taken place, the joint being much swollen and the skin discolored. A thin, sanious fluid, a mixture of blood and synovia, issued from the joint wound, but suppuration had not commenced.

The method of applying the antiseptic dressings was, in the main, that first suggested by Prof. Lister, as reported in Ashhurst's Principles and Practice of Surgery, p. 149, and consisted in covering the joint completely with a compress of sheet lint, of such size as to extend from the middle of the arm to the middle of the forearm, the compress being first saturated with a mixture of carbolic acid (Calvert's solution), one part, to olive oil, four parts. This compress was made thoroughly antiseptic, every portion being soaked in the carbolic acid mixture. A putty, made of carbonate of lime and the carbolic acid mixture, was then spread upon a strong muslin compress, and applied over the first compress, in such manner as to completely envelop the part. A piece of waxed paper was placed over this, and the whole dressing retained in place by a bandage loosely applied. In order to maintain motion in the joint, a Stromeyer's splint was secured to the part, and instructions were given to the resident physician, Dr. Ragan, to change daily the position of the arm by acting upon the joint of the splint.

The patient complained for a short time of slight burning pain, caused by the carbolic acid mixture where it came in contact with the surfaces of the wound. In other respects, he expressed himself as free from pain and discomfort. His condition was very favorable after the application of the dressing, the symptoms of inflammatory action gradually disappearing.

Four days after the dressings were applied, the inner compress of sheet lint was exposed, and on the under surface, near the joint wound, were found evidences of slight suppuration. This compress was not disturbed, but a quantity of the carbolic acid mixture (acid and oil) was poured upon the external surface and rubbed into the meshes of the lint, so as

to saturate it, when a fresh compress covered by the putty was applied with the waxed paper and splint as before. Four days later, the outer dressings were again removed, and it was evident that the suppurative action had increased since the last dressing, there being sufficient pus to saturate the under surface of the lint compress. There was, however, but slight odor. The compress was allowed to remain in place, and again rubbed over with the acid and oil, the outer dressings being replaced as before described, with a change of the putty compress.

In this manner the wound was dressed until the seventeenth day, when *all* of the dressings were removed, and the following conditions were observed: The sheet-lint compress was slightly offensive, the portion in contact with the joint wound being covered with pus which had become dry. Both wounds were entirely filled up, and cicatrization was beginning in each. The motion of the joint was very free, extension being slightly limited; in the forearm, supination and pronation could be accomplished with slight difficulty. The dressings were renewed, with the exception of the compress of putty, and at the expiration of two weeks they were removed finally, the wounds being healed and motion completely established.

The patient received his discharge from the Hospital, and has been able to use his arm in his work; strength and motion were rapidly regained in the joint, and, save the cicatrices which are seen, there is nothing to indicate the grave character of the injury received. There appears to be no impairment of any of the movements belonging to the joint or forearm.

In this case, it will be observed that the antiseptic method was not strictly carried out, owing to the omission to apply to the wounds and wounded surfaces, the strong acid prior to the application of the dressing, as recommended by Prof. Lister. Without

any extended experience in the use of the plan, I hesitated to apply to the interior of the joint the acid in such form; the acid and oil mixture was applied to the wounded surfaces, but not in the manner or quantity recommended.

It is also to be observed that motion of the joint was faithfully practised by means of the splint. The patient was sufficiently intelligent to recognize the great importance of this duty, and contributed without doubt to the successful result by giving attention to this himself. Notwithstanding this combination of treatment, I am still inclined to claim the result as, in a large measure, due to the antiseptic plan. While suppuration occurred, I believe it was limited in extent and duration by the dressing employed. I question whether this result would have been obtained under any other mode of treatment.

CASE

OF

PERIPHERAL NECROSIS OF THE HUMERUS,

INVOLVING THE HEAD AND ENTIRE SHAFT; PRE-EXISTING OSSEOUS ANCHYLOSIS OF THE SHOULDER, AND FIBROUS ANCHYLOSIS OF THE ELBOW; AMPUTATION AT THE SHOULDER-JOINT.

By

J. EWING MEARS, M.D.,

SURGEON TO ST. MARY'S HOSPITAL, ETC.

[Read October 4, 1876.]

The patient, whom I bring before the College to-night, gives the following history:—

P. B., æt. 37, a native of Ireland, and by occupation an accountant; so far as could be ascertained, hereditary disease did not exist in his family. When twelve years of age, after the receipt of an injury and exposure to cold, he was attacked with fever and very painful swellings of the right shoulder-joint and left knee-joint. A month after he was taken sick, matter formed in both joints, and pieces of bone were discharged. From the knee-joint, the pieces were small and not very numerous; but from the shoulder-joint a large piece was discharged, which, according to the description given, resembled the head of the bone.

In six months, the wounds healed, with partial stiffness of the left knee-joint and right elbow-joint, and complete immobility of the right shoulder-joint. From that period until the month of April of this year, he enjoyed very good health,

being able to engage in his duties as accountant and schoolteacher. The stiffness of the knee-joint made him slightly lame, while the movements of the arm were restricted by the immobility of the shoulder-joint.

At this time, he strained his arm in an effort which he made to lift a heavy box. Two days after receipt of the injury, the arm began to swell, and he was attacked with fever, somewhat in the same manner as when he had the first attack twenty-five years before. The swelling continued, and in three or four weeks matter escaped from the arm by a number of openings; small pieces of bone were discharged, and the number of openings increased until there were thirteen in all. During his illness he sustained fracture of the bone, in an effort made to raise his body in bed, the sound produced by the fracture being quite audible.

He entered the hospital on June 15, and was under the care of my colleague, Dr. Grove, who delayed operative interference on account of the patient's condition, transferring him to me on July 1, when I came on duty. The tonic treatment instituted by Dr. Grove was continued until August 1, at which time the patient's condition was thought to be such as to warrant an operation.

With a view to perform excision, if the condition of the bone and tissues were found to be such as to admit of it, an incision was made on the outer side, beginning about midway between the shoulder-joint and the insertion of the deltoid muscle, and extending below the middle of the arm. This incision permitted an inspection of the tissues, which were found to be so changed by inflammatory action as to forbid any attempt to save the limb. Amputation at the shoulder-joint was then determined upon, and performed by Dupuytren's method. On exposing the joint, firm, bony anchylosis was found to exist. Section of the bone, as near to the joint as possible, was made with the saw, the remaining portion being removed by the forceps and chisel.

The recovery of the patient was as rapid as could be expected for one in his condition; it was somewhat retarded

by the formation of an abscess in the lumbar region, from which two pints of gelatinous pus were removed by the aspirator. The union of the flaps occurred with but slight suppuration.

A dissection of the limb was made after removal, and the soft structures were found much changed by the extensive suppuration which had occurred; they were perforated by numerous sinuses lined with unhealthy granulations. The periosteum was at some points destroyed; at others, indurated and much thickened; in a few places, there were slight evidences that its osteogenetic function still existed. At the time of the operation it was ascertained that bony anchylosis existed at the joint, and that, as stated by the patient, the head of the bone had been separated and discharged during the first attack of illness; the upper portion of the bone, corresponding to that embraced between the tuberosities and the insertion of the pectoralis major muscle, was but slightly affected, although there were evidences that it had been attacked in the previous disease; the compact tissue was removed, leaving the central cancellated portion, which was somewhat softened, and, when compressed by the pliers, exuded a frothy fluid. Below the surgical neck, the shaft of the bone was found to be in a condition of extensive peripheral necrosis, giving it a worm-eaten appearance; this condition extended to the articulating surface of the bone, but did not involve it. At about the junction of the upper with the middle third of the bone, there was a fracture, in which fibrous union had occurred. There was no evidence of an attempt on the part of nature to obtain any osseous union; the ends of the bone were in apposition, and the surfaces were smooth. The appearance of the bone is shown in the accompanying illustration (page 20).

It would appear from the history given by the patient that, in all probability, the attack of illness which occurred at twelve years of age, was arthritis, following contusion of the joints and exposure to

cold, the former being produced by a fall from a horse. In the shoulder-joint, the disease was very extensive, producing complete destruction of the articulation, and obliterating its structures. The epiphysis, being separate at that time, was easily detached, and removed by the suppurative action. The knee-joint was not involved to any great extent, and the fibrous anchylosis of the elbow-joint was, without doubt, the result of disuse, assisted by the contraction and adhesion of the adjacent muscles and tendons.

The second attack of illness, that occurring in April of this year, was evidently one of *diffuse periostitis*, following the injury sustained in lifting a heavy weight. A condition of the periosteum may have existed, as the result of the previous disease, which rendered it susceptible to an attack of this nature.

With regard to the treatment of these cases, the most energetic means must be employed in the first stage of the inflammatory action—free incisions to relieve the tension of the periosteum, and to deplete it, and also to permit free escape of the pus should this form; at the same time constitutional remedies for the purpose of maintaining the strength of the patient should be resorted to. When extensive destruction of the bone ensues, amputation must be performed, providing the condition of the patient will admit of it.

THE POST-MORTEM IMBIBITION OF POISONS: MORE ESPECIALLY IN ITS MEDICO-LEGAL RELATIONS.

By

JOHN J. REESE, M.D.,

PROFESSOR OF MEDICAL JURISPRUDENCE AND TOXICOLOGY IN THE UNIVERSITY OF PENNSYLVANIA, PHYSICIAN TO ST. JOSEPH'S HOSPITAL, AND TO THE GYNÆCOLOGICAL HOSPITAL AND INFIRMARY FOR DISEASES OF CHILDREN.

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In a late number of the Philadelphia Medical Times (Aug. 19, 1876), the very pertinent question is proposed by a correspondent, whether the embalming a dead body by means of a solution of arsenic, injected into the arteries, might not be the means of arresting the arm of justice in a case where the death had been occasioned by poisoning with arsenic? For, as the querist very properly observes, "the art of the toxicologist would be of no avail" under the circumstances, in detecting the poison in the tissues after death, inasmuch as its presence there could satisfactorily be ascribed to imbibition, or soaking of the embalming fluid employed.

The above query has suggested to me the propriety of examining into this subject a little farther; and its consideration has seemed to be of sufficient interest and importance to justify me in bringing it to your notice, very briefly, this evening.

The *post-mortem imbibition of poisons*, or the possibility of the absorption of a poison into the body after death, is a question of very considerable medico-legal importance, as will be seen on a slight examination. In order properly to appreciate its toxicological bearing, it will be requisite to refer to two or three preliminary propositions.

The first of these is that a poison is *active*, *i.e.*, is producing its deleterious effects, only whilst it is circulating in the blood; or, to express it differently, the actual, potential *dose* of the poison is that which is circulating, dissolved, through the bloodvessels. All the balance of the poison, however large the quantity may be, remaining either in the stomach, or separated by, and contained in, the organs and tissues, is totally inert *for the time being;* so that if an ounce of arsenic, or of morphia, for example, be swallowed, the real toxic effect is due solely to the comparatively minute quantity—a few grains of either—which has undergone solution, and has been absorbed into the blood. There is one exception only to the above law, in the case of the true corrosives, such as the mineral acids and the caustic alkalies; these, as is well known, produce a direct, destructive action upon the tissue with which they come in contact, by virtue of their chemical affinities.

Another proposition, to which I must refer, is the now well-ascertained fact that, no sooner does the absorbed poison find its way into the circulation, than provision is made for its speedy separation and elimination through the medium of the different organs and tissues, more especially the liver, kidney, spleen, heart, and sometimes the brain and spinal cord; and

many of these noxious agents—the mineral poisons particularly—may be readily discovered, soon after their administration, in the different secretions of the body, notably in the urine, but likewise in the bile, saliva and sweat.

It is farther to be remembered that, when the absorbed poison has thus been separated by the different organs, it is not retained by them for an indefinite length of time; but it disappears, or is reabsorbed, in a period varying both according to the kind of poison employed, and also according to the organ involved. For the knowledge of this important toxicological fact, we are indebted chiefly to the researches of the two Orfilas, and likewise to Prof. A. S. Taylor. Thus, to take a single illustration, *arsenic* makes its appearance in the human liver, and has there been detected by analysis, in four hours after that poison has been swallowed. Doubtless, it reaches the liver much earlier than this, but as death from arsenical poisoning is not apt to occur in man within a shorter period of time than four hours, no opportunity has hitherto occurred of proving the fact in the human subject. Some experiments on the lower animals go to show that arsenic is very completely diffused throughout the body in one hour and a half after being introduced.

The human liver acquires its maximum of saturation, in the case of arsenic, in about fifteen hours after the ingestion of this poison; and the quantity thus separated, and capable of demonstration by the analyst, rarely ever exceeds two grains. After this period, the amount discoverable gradually diminishes; thus in ten days it was found to be only 1.5 gr.; in

fourteen days, 0.17 gr.; and in seventeen days, none at all: it had by that time been entirely reabsorbed. (Taylor.)

The generally received opinion is, in relation to arsenic, that, if an individual poisoned by this substance survives for sixteen or eighteen days, and then dies, there will be little, if any, probability of detecting the poison in the liver after death. But, inasmuch as the vast majority of such cases of poisoning prove fatal within eighteen hours, the toxicologist has nearly always the opportunity (which he should never neglect) of discovering the metal in the liver and other organs of the body.

This leads me to allude to a third proposition, which naturally follows from the two preceding ones: viz., that one of the very strongest proofs of death by poison, and the one which especially engages the skill of the analyst, is, *not* the detection of the alleged poison in the stomach (since there may be always a possibility of its having been designedly introduced there after death), but its discovery in the absorbed state, in the liver, kidneys, spleen and other organs; the presumption amounting almost to an absolute certainty that the noxious substance thus detected was administered during life, and while the blood-circulation was going on.

Keeping then in mind the important medico-legal fact that the discovery by the toxicologist of the absorbed poison in the tissues and organs of the deceased is regarded as irrefragable proof that the said poison was administered *during life*, it becomes a most interesting and important subject of inquiry: is it possible for a poison to get access within a

human body *after death*, and produce appearances in that body similar to, if not identical with, those resulting from swallowing the same poison during life?

It will be obvious, on a moment's reflection, that if the affirmative of this proposition can be maintained, a very strong point in legal medicine will be made; since what has heretofore been regarded as one of the firmest pillars of evidence for the prosecution, in certain cases of poisoning, will be shaken, and a powerful weapon will be placed in the hands of the defence, which may be wielded by a skilful counsel either for good or evil.

Let us now, for a few moments, examine what are the actual facts of the case. There are only two methods by which a dead human body can receive a poison into itself: one, the *accidental*, as when after burial in a soil that may chance to be impregnated with a poisonous material, in process of time, owing to the decay and disintegration of the coffin, the animal remains come into immediate contact with the poisoned soil; the other method, the *intentional*, where the poison in solution has been purposely introduced through the œsophagus into the stomach, or into the rectum, or hypodermically into the cellular tissue, or finally into the bloodvessels (as in the process of embalming, so called).

As regards the former of these methods, very little need now be said. Prior to the time of Orfila, the opinion generally prevailed that the contamination of a dead body, more especially by arsenic, in the soil of a cemetery, was quite possible; hence we find that this idea was very often urged as a plausible ground

of defence, in cases of alleged poisoning by arsenic. This theory has, however, long since been abandoned, for the very sufficient reason that, in the few actual cases in which arsenic has been discovered in cemetery soils, it has invariably existed in the *insoluble* state—generally in combination with either iron or lime. Indeed it cannot be extracted from such soils even by boiling water, but the agency of hydrochloric acid is required to render it soluble: consequently, it is impossible that arsenic should be capable of transudation from the soil into a dead body. No sane man would, at the present day, dream of advancing such a plea in the defence of a case of alleged poisoning by arsenic.

But the second method by which a dead body may be impregnated with a poison, and which I have named the *intentional*, is by no means so easily disposed of. The question here is, whether it is possible for a poison existing in the stomach at the time of death (and the death not necessarily due to the poison), or introduced into it after death, to be diffused throughout the body by imbibition, so as to be discovered *in the organs* by chemical analysis? The all-important point, you will observe, is the discovery of the poison *in the organs*, under these circumstances. My reply to this question is that such a result is most certainly possible; and that it is easily explained on the well-known physical law of *osmosis*.

Orfila was, I believe, the first to actually demonstrate the fact upon dead human and animal bodies, with solutions of arsenic, corrosive sublimate, antimony, and copper. His experiments clearly show that, when these solutions are injected into the stomach or

rectum, after death, an imbibition does actually take place slowly and gradually through the coats of these viscera into the neighboring organs; affecting first and chiefly (as might naturally be expected) those viscera which are in immediate juxtaposition with the stomach or rectum, and for the most part limiting its action to the nearest surface of the organ. Thus, for example, a poisonous solution, placed in the stomach after death, would gradually escape through its walls by osmosis, affecting first the contignous viscera, as the lower surface of the liver, the left under side of the diaphragm, the right side of the spleen, the transverse colon, the omentum, and, still later, the lower lobes of the lungs, the upper surface of the diaphragm, the kidneys, and other viscera.

It certainly needs no argument to show how easily the legal physician might be deceived in a case of this character, mistaking the effects of a simple imbibition or soaking of a poisonous solution which had been introduced designedly into the stomach or rectum *after* death, and of course totally disconnected with the death; and very naturally attribute the results to poison really absorbed during life, and regard them therefore as affording unquestionable proof of the alleged crime. Orfila himself puts this matter very pointedly: "Suppose," says he, "that some wretch, with the design of accusing an innocent person of the crime of poisoning, should introduce into the digestive canal of the dead body a poisonous solution, which would afterwards penetrate by imbibition even to the remotest organs, from which it would be subsequently extracted by the experts, and

would lead them to the conclusion that they were dealing with a veritable case of poisoning!"

I suppose that enough has been said to demonstrate at least the fact that the post-mortem imbibition of poisons is not only a possible thing, but also one of comparatively easy accomplishment. Nevertheless, in all the annals of poisoning, which have been marked by so many subtle and crafty machinations, including the mysteries of the secret poisonings of a Toffana, a Borgia, and a Brinvilliers, no mention whatever is made of any attempt of this kind, by which this odious crime has been sought to be fastened upon an innocent person. Indeed, while Orfila fully admits the possibility of the crime, he distinctly asserts that, to his knowledge, "such a refinement of human wickedness had never been brought before the tribunals of any country," and Sir Robert Christison remarks: "Although I have never been able to find any authentic instance of so horrible an act of ingenuity having been perpetrated, it must nevertheless be allowed to be quite possible."

About five years ago, a remarkable case of alleged poisoning by arsenic occurred in one of our Western States, in which the suspicions were exceedingly strong that the poison had been thus designedly introduced after death, for sinister purposes. The deceased was an aged man, who had been treated in his last illness for phthisis; his physician subsequently testifying to his having died of this disease, and to his having presented none of the symptoms of arsenical poisoning before death. The body had been buried some three or four years, during all of which

long interval of time no suspicion of foul play appears to have been entertained. In the meanwhile the widow married again; after which, for reasons not known to me, but believed to be dependent on questions of inheritance of the property of the deceased, the suspicion of poisoning was bruited about, the woman was accused of the crime, and the body was exhumed for judicial examination. The autopsy revealed a remarkable state of preservation of the body—a circumstance of itself well calculated to sustain the suspicion of arsenical poisoning; and, as if to remove all shadow of a doubt, this poison was actually detected in the stomach and liver by a distinguished chemist.

Now, one would say that here, surely, was a very strong case: could there be any doubt about it? There was the remarkable preservation of the body for over three years, evidently due, as it seemed, to some antiseptic agent; and then, farther, there was the actual discovery of the deadly substance, not only in the stomach but in the liver—showing, it might be urged, that the poison must have been absorbed during the life of the individual.

What then was the ground of the defence to refute these two strong points of the prosecution? First, as to the preservation of the body, it is well known that many instances of remarkable resistance to putrefaction occur in dead bodies without the presence of arsenic; whilst it is equally well established that arsenic does not always prevent even the rapid decomposition of a body. So that this point amounts merely to a suspicion, and to nothing more. Moreover, in this connection, the liver is stated to have

been "very much broken down." Now, inasmuch as there seems to be, so to speak, a remarkable affinity for arsenic by the liver (a large part of absorbed arsenic being invariably found in this organ), we should naturally expect that the conservative influence of the antiseptic would have been exerted specially upon the liver, if the arsenic had, in very truth, been absorbed into it during life. In my own experience, in such cases, the liver has always exhibited a remarkable degree of firmness even years after death. I therefore regard the "broken down" condition of the liver in this case as a circumstance making against the idea of ante-mortem poisoning.

Another significant fact is that no mention is made of the discovery of any yellow sulphide of arsenic in the disinterred body. It is well known that when death has been occasioned by ordinary arsenious acid, this substance becomes changed into the yellow sulphide, in the process of decomposition, through the agency of the evolved sulphuretted hydrogen gas. This conversion is very distinct, and may always be looked for, if a sufficient length of time be allowed; and the longer the interval, the more certain and complete the change. Certainly, then, the absence of this appearance after a burial of over three years, is a very significant circumstance for the defence.

At all events, the defence planted itself firmly on the ground that the poison had been designedly introduced into the body, not very long before its disinterment, with the view of fastening the guilt upon the woman, and thus diverting the estate of the deceased into another channel. I have learned indeed that this was the opinion of persons most

conversant with the facts of the case. There was certainly a strong motive for this sinister and secret act, and there was sufficient opportunity for accomplishing it, as the body had been buried in a vault; and, as we all know, *motive* and *opportunity* constitute very strong circumstantial proof. A most significant fact, moreover, in connection with this case, is that it had only a preliminary hearing: it was abandoned by the prosecution before coming up for final trial. This, I presume, would hardly have been the issue if the prosecution had felt sure of its ground. As this case is the only one of the kind on record, so far as I know, we must regard it as of considerable medico-legal importance as a leading one in this line. The only regret is that many of the particulars are deficient; and I have found it impossible to get at the details.

Impressed with the importance of this subject to the legal physician, I made the suggestion to the class in Medical Jurisprudence at the University, at its last session, that some of the students should take up the topic as the basis of an experimental thesis, with a view of securing the annual prize instituted by one of the Alumni Societies. I feel a gratification in saying that one member of the class did make a very complete series of experiments in this line, extending over a period of three months, upon the bodies of dogs. At my suggestion, he confined his experiments, for the time, to three poisons—arsenic, tartar emetic, and corrosive sublimate, injecting strong solutions of these mineral substances into the stomachs of the dead animals; then burying them beneath the ground; and disinterring them at different

periods of time, so as to note the difference of result, as dependent on the length of time of burial.

From this very interesting essay of Dr. George McCracken, of this city, which I learn will be published, I extract the following brief particulars bearing on our subject:—

The periods of interment were respectively twenty-one, thirty-seven, forty-four, and fifty-nine days.

After twenty-one days' burial, in the case of all the three poisonous solutions, on opening the abdomen of the animal, the characteristic colored spots of the respective sulphides were observed on the spleen, the under surface of the liver, and the portion of peritoneum posterior to the stomach—*yellow*-colored in the case of arsenic; *red* in the case of antimony; and *black* in the case of mercury. Each of these metals was likewise discovered by chemical analysis in the liver, spleen, and left kidney; the order for the greatest amount of the poison detected being first in the spleen, next in that part of the liver nearest the stomach, then in the left kidney, then in that part of the liver farthest from the stomach, and none in the right kidney.

After forty-four days' burial, the deposit of the different sulphides was found to be much more decided in all three cases, being noticed on the upper as well as on the lower surface of the liver, together with the spleen (as in the first experiment); also over the intestines, the omentum, and the kidneys; and, in the case of the arsenic, extending as low down as the fundus of the bladder. By chemical analysis, the poisons were detected in the spleen, liver, and both kidneys.

After fifty-nine days' burial, the results were found to be very similar to those last detailed, only more decided, both as regards the quantity deposited on the various organs, in the form of sulphides, and that discovered by chemical analysis.

From his experiments, thus epitomized by me, Dr. McCracken deduces the two following conclusions: First, If a solution of either arsenious acid, tartar emetic, or corrosive sublimate (and this is doubtless true of all other metallic solutions, and presumably true of the organic poisons likewise), be injected into a dog's stomach after death, it passes by osmosis through the coats of this organ into the adjoining viscera, and may be discovered in three weeks (his earliest experiment), by chemical analysis, in the spleen, the liver, and the left kidney; but not in the right kidney prior to the fifth week.

I myself think it highly probable that the osmotic action occurs even earlier than this, and that an earlier experiment would have demonstrated the presence of the poisons, in at least some of the contiguous viscera, within a still shorter period.

The second conclusion arrived at is that the arsenic solution penetrates through the stomach more rapidly and completely than the other two substances employed. I am pleased to learn that Dr. McCracken intends to prosecute his experiments farther, upon human bodies, and with various poisonous solutions; but I cannot doubt that the results must be similar to those I have just detailed, inasmuch as these are dependent upon a great physical law, which operates in a precisely similar manner upon the dead tissues of man and those of the lower animals.

In summing up this whole matter, the really important practical question to be settled is this—and it is one of vital medico-legal interest: Is it

possible, in an unknown case of poisoning, where the whole issue depends on the chemical discovery of the poison in the tissues of the body, to distinguish between poison really *absorbed* during life, and that which has been merely *imbibed* after death? Of course, I put out of the question all the other collateral evidences, such as the symptoms before death, the post-mortem lesions, together with the moral or circumstantial proofs in the case. Can the distinction be positively made?

My reply is this: If I had the opportunity to make a careful comparative analysis of the *interior* of the organ, the liver for example, with the exterior of the same organ—especially if the examination were made not long after death—and I discovered the poison in the interior as well as on the external part of the organ, I should be disposed to regard it as evidence of really absorbed poison, inasmuch as a substance absorbed into the blood during life, would unquestionably find its way through the route of the circulation into the most interior recesses of an organ, and would not be confined to its exterior merely; whereas, if it had simply leaked out of the stomach or rectum after death, it would be far more likely to be discovered on, or near the surface of, the contiguous viscera, than deep down in the interior of their structure.

But, on the other hand, if the poison were found on the exterior of the organs only, and not in their interior, after a careful research, I should regard it as a true case of post-mortem imbibition. Or, at least, presuming it to be such, I should direct my investigations accordingly.

Some recent experiments of M. Scolosuboff, of Moscow (Archives de Physiologie, No. 5, Août et Septembre, 1875), go to show that, in dogs and rabbits poisoned by arsenic, this substance is deposited in the brain and spinal marrow in far larger quantities than in the liver and other organs. Comparative analyses of equal weights of muscle, liver, brain, and spinal marrow, taken from a dog that had been fed on arsenic for five weeks, demonstrated that the brain and spinal marrow contained thirty-six and thirty-seven times the amount found in the muscles, and nearly four times that discovered in the liver. In acute poisoning (the animal dying in 17 hours), the brain was found to contain rather more than the spinal cord.

From the close resemblance of some of the symptoms of arsenical poisoning—*e. g.*, the local paralysis and muscular atrophy—with those produced by lead, and from the fact that this latter metal had been discovered, after death, in the great nerve centres, it might have been inferred that arsenic would also be deposited in these organs; but M. Scolosuboff is, I believe, the first to have made the actual demonstration in the lower animals. I am not aware that it has yet been verified in man, although there seems good reason to believe that it will equally apply to the human subject. Should this prove to be the fact, we shall be in possession of a positive and unequivocal chemical method of distinguishing between ante-mortem and post-mortem poisoning by arsenic (and probably by other metals), by the detection of the poison in the brain and spinal cord; since it is scarcely conceivable that a poison, introduced into a body after death, could penetrate by imbibition within

the cavity of the cranium or spinal column. At all events, it will henceforth become the duty of the expert, in all doubtful cases, to extend his researches for the poison to the brain and spinal cord.

After all, however, cases may occur of a complicated character, which on the trial may give rise to painful doubts, and in which, as I before remarked, a skilful advocate might employ these ascertained facts with considerable ingenuity, either for or against the cause of truth. As the result of some experience and considerable reflection on this subject, it is my deliberate opinion that, in a trial for the capital crime of poisoning, the defence has the undoubted right to demand on the part of the chemical expert who has conducted the analysis of the body of the deceased, not merely proof of the detection of the alleged poison in the stomach, nor even proof of its discovery in the organs and tissues of the body, but, even farther than this, evidence, clear and unmistakable, of its detection in the *interior* of these organs; and farther still—from what we now know—in the brain and spinal cord. Of course I am aware that the guilt of the prisoner may sometimes be established quite independently of the chemical analysis, but with that point we have nothing to do at present; I am now discussing this chemical evidence exclusively; and I would here repeat, what I have elsewhere asserted, that, inasmuch as upon this very chemical evidence may be suspended the momentous issue of the life or death of a fellow being, nothing should be accepted from the expert as testimony, but a demonstration of his discovery, so clear, so searching, and so unequivocal, as to suggest to the mind no shadow of a doubt.

ON THE

INTERNAL ADMINISTRATION OF NITRATE OF SILVER, AND ON THE OCCURRENCE OF A BLUE LINE UPON THE GUMS AS THE EARLIEST SIGN OF ARGYRIA.

By

WILLIAM PEPPER, A.M., M.D.,

PROFESSOR OF CLINICAL MEDICINE IN THE UNIVERSITY OF PENNSYLVANIA.

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It is of course not within the province of the present article to discuss the very numerous and varied conditions in which the internal administration of nitrate of silver is strongly indicated. Suffice it to say that there are few remedial agents whose therapeutic value and range of application are equally extensive. This is not surprising when it is remembered that nitrate of silver possesses a peculiarly valuable local action in addition to its power of directly affecting the nervous system by absorption. The topical action of nitrate of silver, given internally, even in small doses, may be said to extend throughout the entire gastro-intestinal tract, with, perhaps, the exception of the rectum. In addition to this, it appears to me to be established by clinical evidence that a certain amount of influence is produced on the continuation of the duodenal mucous membrane into the common hepatic duct, and possibly into the pancreatic duct. This local action is in part that of an astringent, but

in addition it presents a peculiar, alterative character. Its power of influencing nutrition enables the nitrate of silver to modify the morbid action of ulcers of the stomach or intestinal canal, and to cause the reduction of chronic inflammatory thickenings of the mucous membrane, and of hypertrophies of its glandular apparatus. Associated with this is its power of allaying morbid irritability of the nerves of the mucous membrane, and of influencing the secretions.

The exact form which nitrate of silver assumes after entering the stomach is not known, and probably is not the same in all cases. It is evident, however, that it cannot long retain its integrity. Probably it is sometimes converted into a chloride, though more frequently it would seem to unite with the albuminous peptones, forming a compound which does not coagulate albumen, and which is readily dissolved in albuminous liquids. In this form it may easily be understood how, even when given in minute doses, if frequently repeated and continued for a considerable time, it may produce a powerful action throughout the entire extent of the mucous tract before mentioned.

Among the special morbid conditions for which it has been recommended, on account of its local action, I would especially mention the following, based upon my own personal experience: in the stomach—gastric ulcer, chronic catarrhal gastritis, especially with frequent vomiting or with pain dependent upon morbid irritability of the nerves of the mucous membrane; in the intestinal canal—cases of follicular (catarrhal) enteritis, both in the adult and in the child, subacute and chronic dysentery, chronic typhlitis, and tubercular diarrhœa. I have also lately obtained remarkable

results from the administration of nitrate of silver in cases of gastro-hepatic catarrh, with or without jaundice from occlusion of the ducts by tumefaction of the mucous membrane. Recent observation has, moreover, led me to believe that the administration of this drug in typhoid fever, as originally recommended by Bondin in 1836, is a very valuable element of treatment in this important affection. In addition to all of these must be mentioned the great value of injections of nitrate of silver in many forms of rectal disease.

With regard to its absorption and effects upon the general system, the evidence is no less satisfactory. The fact of its absorption does not indeed admit of doubt. Apart from the positive therapeutic effects observed, there is the actual proof of the detection of silver in various internal organs, and of its deposit in the skin after its long-continued administration. Given thus internally, the chief action of the salt would seem to be upon the nervous centres, whose nutrition it possesses the power of modifying to a considerable degree, though the precise mode in which this is accomplished is still uncertain. The diseases for which nitrate of silver is used in view of its constitutional effects are chiefly affections of the nervous system, such as epilepsy, chronic meningitis, and sclerosis of the spinal cord.

In regard to a remedy of such value and wide application, it is important to consider what difficulties attend its administration, and how they may best be overcome. The first difficulty which suggests itself is connected with the local irritant action which nitrate of silver occasionally produces. This, however, can always be avoided by giving the remedy in pill form,

and by graduating the dose carefully in accordance with the sensibility of the stomach. Thus, my own rule is to begin the administration of this drug in doses of from one-sixth to one-fourth of a grain three times a day, to give it in pilular form, and soon after a little light food has been taken. If necessary, its irritant action may be still further guarded against by combining with it a small quantity of opium.

It is usually stated that when the local action on the alimentary canal is desired, the drug should be given one or two hours before meals, on an empty stomach; while when the constitutional effect is desired, the pill should be given after meals, during the process of digestion. In most cases where nitrate of silver is given for its local action, it is necessary to employ a diet carefully restricted, both as to quantity and character; and it has seemed to me that the good effects of the remedy are not interfered with by its being administered half an hour to an hour after a very light meal, especially of a fluid character, such as skimmed milk or broth. It is certain that the danger of causing gastric irritation is much lessened when this drug is thus given, instead of being given on an empty stomach before meals. In cases where the diet allowed is not so rigidly restricted, and considerable quantities of food are taken at each meal, and yet the administration of the silver on an empty stomach causes irritation, I have found it useful to give a few spoonfuls of light arrow-root a couple of hours before the regular meal, and to administer the silver immediately after this.

The second great difficulty attaching to the internal administration of nitrate of silver is the tendency to

discoloration of the skin by the deposit of the silver in its tissues. This discoloration of the skin has long been known. Fourcroy (Médecine éclairée par les Sciences Physiques, tome i. p. 342) was among the first to call attention to it; Dr. I. A. Albers, of Bremen, communicated a case to the Royal Medico-Chirurgical Society of London, in 1815 (Med.-Chir. Transactions, vol. vii. p. 284); and since that time numerous cases have been placed upon record.

The skin first presents a peculiar, bluish-slate color which may deepen until the patient acquires a hue of the deepest lividity. In some cases, where the discoloration has become very intense, the color is described as bronzed. This discoloration is usually said to be deepest in parts exposed to the light, as the face and hands. The whole surface is, however, involved, and in severe cases the sclerotic coats of the eyes, the mucous membrane of the mouth, and even the internal organs, as the liver, spleen, and kidneys, have been found to be discolored. So grave is the danger of inducing this condition, and so universally is it regarded as a serious contra-indication to the use of this valuable remedy, that it is important to consider somewhat more in detail (1) the cause of this discoloration; (2) the shortest period of administration and the smallest quantity of the nitrate of silver after which it has been known to occur, and (3) the best mode of avoiding this unfortunate result.

In regard to the first of these points, the fact that the discoloration is not limited to the external surface, but affects also the internal organs, goes far to prove that it is not due to the chloride of silver, but to a

deposit of the metal itself or its oxide, and this view is further confirmed by chemical examination.

The amount of silver in the tissues required to produce this discoloration is extremely small, thus H. C. Wood quotes the analysis of the organs in a case of Versmanns (Virchow's Archiv, xvii., 1859), who in 14.1 grammes of dried liver found only 0.068 gramme of metallic silver (0.047 per cent.), and in 8.6 grammes of dried kidney, 0.053 gramme (0.061 per cent.).

The smallest amount which has ever been known to produce general discoloration is difficult to determine. It has been known to follow the use of one hundred and twenty grains, given in the dose of half a grain daily for eight months consecutively; while again, in a case reported by Bertini (De usu interno preparationum argenti, Thesis inauguralis. Geneva, 1814), thirty-three and a half drachms were taken in a period of two years and a half, averaging two and a half grains daily. These cases may perhaps be taken as representing about the extremes; but in the latter instance it is not stated how long the drug had been taken before the discoloration was first noticed, nor is it possible, of course, to form any idea of how much of the great amount given passed through the alimentary canal unabsorbed.

With regard to the length of time necessary for the development of the discoloration, Dr. James Johnson asserts that there is no case of discoloration on record in which the use of the medicine had not been continued beyond three months. Much more frequently, however, when argyria, or discoloration of the skin by silver, is produced, it will be found that the patient has continued the use of the remedy with or without

the advice of the physician for a much longer period of time than the above, usually eighteen months or over. It is interesting to note, in this connection, that so large a proportion of the recorded cases of argyria has occurred in epileptics. It is well known that, before the discovery of the peculiar powers of the bromides, the long-continued use of large doses of nitrate of silver was strongly recommended in this disease.

Another peculiarity connected with argyria, according to some observers, is that the discoloration continues to deepen for some time after the use of the remedy has been suspended. It is even asserted (Sigmund, Lancet, March 31, 1838) that in some instances the discoloration does not occur until some months after the remedy has been discontinued, but this appears to me very improbable, and certainly needs confirmation.

In regard to the mode of avoiding this unfortunate result, various suggestions have been made. Thompson proposed the simultaneous administration of nitric acid, with a view of preventing the conversion of the nitrate into the chloride. This, however, has been shown, both theoretically and practically, to be useless. The majority of authors content themselves with recommending that the administration of the silver salt should be suspended after a period which, according to different authorities, varies from three weeks to three months. It is evident, however, that this is altogether too vague to be depended upon. It would be more valuable could some safe limit as to the quantity to be used be determined. My own rule, for a number of years, has been to continue its administration in doses

of from one-fifth to one-third of a grain thrice daily until thirty grains have been taken; to then suspend it for one month; after that interval to resume and continue its administration until fifteen grains more have been taken; and again to withdraw it either permanently or for another month, to be then followed by a final course of fifteen grains, making sixty grains in all. One great defect, however, of such rules, is that they only fix a minimum dose without reference to the individual features of the case, and, of course, without any accurate knowledge of the proportion of the remedy absorbed in different instances. Thus, among other considerations which deserve attention, is the weight of the patient. In the case of nitrate of silver, as of many other drugs capable of producing an effect on the whole body, it cannot be doubted that one of the factors, in determining the amount that can be tolerated in any given case, is the weight of the subject. But when we consider the extreme value of this remedy in certain conditions, it becomes evident that what we need, is a rule which will enable us to administer, in any given case, the maximum dose consistent with entire safety. It is with the hope of establishing such a rule that I venture to bring forward the results of certain recent observations that I have made, which go to show that *the earliest sign of argyria, occurring before there is any danger of the slightest discoloration of the skin, is a peculiar blue line on the gums.*

The following cases are given in illustration of this position. It may be premised that crystals of nitrate of silver were used exclusively in compounding the

pills administered in these cases, and that consequently there is no chance of any lead having been admixed. This is mentioned because the nitrate of lead is sometimes associated with the nitrate of silver in the form of stick.

Case I. *Chronic Gastro-hepatic Catarrh.*—Mrs. W., æt. 60, of unusually large frame; had suffered from deep jaundice of six months' standing, subject to marked variations in intensity, and associated with frequent spells of hepatic colic from closure of the common duct owing to swelling of the mucous membrane. Treated by nitrate of silver and Carlsbad water, with restricted diet. Complete cure at end of eight months. About sixty grains of nitrate of silver were given in all, in doses of one-sixth to one-fourth of a grain three times a day in pill form. Thirty grains were given at first, then, after an interval of a month, fifteen grains more; and later, after a longer interval, fifteen grains more. No trace of discoloration of the skin, and no line on the gums.

Case II. *Chronic Gastro-hepatic Catarrh.*—Mrs. G., æt. 58, of medium size, was desperately ill for many months, with deep jaundice, clay-colored stools, irregular chills and fever, frequent vomiting, extreme emaciation, intense attacks of hepatic colic: no gall-stone. Complete cure resulted from the use of nitrate of silver, given in doses of one-fourth of a grain with a small dose of opium three times a day. About fifty grains were given. At the close of this time a blue line made its appearance on the lower gum corresponding to the two incisors, canine, and first premolar teeth on right side of jaw. The line was close to junction with teeth, and closely resembled the lead line. The nitrate of silver was dispensed in form of crystals.

Case III. *Gastric Ulcers.*—Mrs. B., æt. 30, rather small-sized, slender woman. Gastric ulcer, profuse hemorrhage, intense gastralgia and heartburn. After controlling the hemorrhage, nitrate of silver was given in pill form, in doses of one-fourth of a grain, thrice daily, taken immediately after

taking small quantities of milk. Rapid improvement. About forty-five grains of nitrate of silver were taken in all, extending over a period of about two months. One month afterwards, examination of her mouth showed a very marked blue line on lower gum, corresponding to the five or six teeth to the right of the median line. The nitrate of silver was given in the form of crystals.

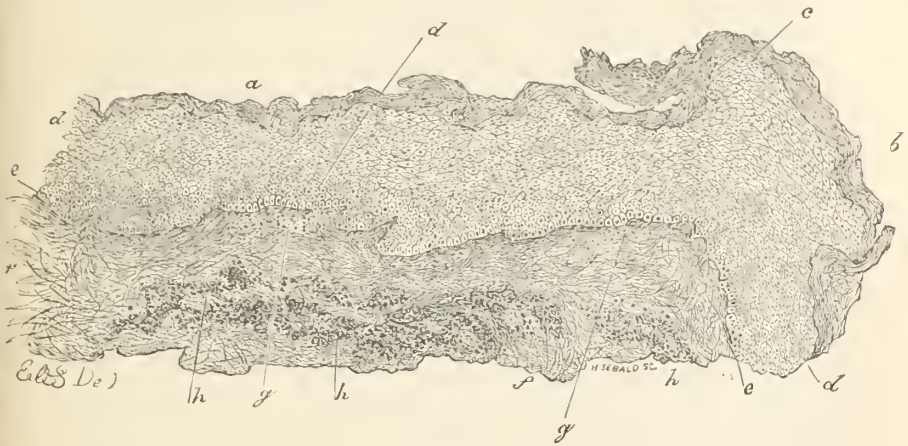
Case IV. *Chronic Perityphlitis.*—H. W., æt. 25, suffering with chronic perityphlitis from a strain; seen in consultation with Dr. Santee; was treated with restricted diet and nitrate of silver in pill form. About fifty grains in all were taken between July 5, 1876, and October 1, 1876. After the above period it was discovered that there was a very marked blue line on the gums, closely resembling the blue line of lead-poisoning.[1] This chiefly affected the lower jaw, where it corresponded to about six or seven teeth counting to the right from the left lateral incisor.

The line was quite broad, in places nearly one line. On the upper jaw a slightly developed line was noticed, corresponding to the right lateral incisor, canine, and first premolar. In addition to this, there was some excessive pigmentation of the mucous membrane of the lower lip near its junction with the gum. There was no trace of discoloration of the skin. A small piece of the discolored portion of the gum was cut out and placed in the hands of Dr. E. O. Shakespeare, who has kindly furnished the following report of the microscopical and micro-chemical examination of the specimen:—

The small portion of tissue from the gum was hardened in dilute alcohol, and, when of sufficient consistence for cutting, the piece measured in length $\frac{1}{2}\frac{1}{5}$ inch, in breadth $\frac{1}{4}\frac{1}{0}$ inch, and in thickness $\frac{1}{1}\frac{1}{0}$ inch. It was imbedded in paraffin in order to make thin sections from it. Cuts were intended to be made vertical to the upper edge of the gum and to its anterior epithelial surface, but, when placed under the mi-

[1] The painting, which was exhibited in connection with this case, has not been reproduced, on account of this close resemblance between the silver line and the familiar blue lead-line on the gums.

croscope, they were found to be somewhat oblique to the latter surface. Some of these sections were stained with carmine and mounted in balsam, others were mounted in the same medium unstained. The accompanying drawing, by Dr. Shakespeare, represents one of the former, enlarged one hundred and fifty diameters. (See wood-cut.)



Section of gum, showing supposed silver deposit along course of vessels.—*a*. Anterior epithelial surface. *b*. Upper edge of gum. *c*. Corneous layer of epithelium. *d*. Rete mucosum. *e*. Layer of cylindrical cells. *f*. Fibrous substance of gum. *g*. A few black granules deposited along vessels of papillæ. *h*. Dark granular deposit covering and occupying the vessel walls.

Under the microscope, the stained section exhibited the following appearances: The epithelial layer of the gum was a little thickened. The layer of cylindrical cells covering the papillary surface was so entirely free from pigment as to lead to the assumption that the patient was very light-complexioned. The outlines of these palisade-like cells were slightly indistinct, the contents finely granular, and the nuclei large and deeply stained. The cells of the rete Malpighii presented large, deeply stained nuclei, granular contents and ill defined outlines. The squamous epithelium near the corneous layer, which was not increased much in thickness, also showed nuclei very distinct and well stained. Through-

out the sub-epithelial, dense, fibrous tissue of the gum, as well in the papillæ as in the deeper portions, but particularly in the latter, was to be observed a very considerable and diffuse cellular infiltration. This cellular infiltration has not been represented in the drawing, but in other respects the latter is as nearly as possible an accurate copy of the microscopic picture presented by the section from which it was made by aid of the camera. In the deeper portion of the fibrous tissue of the gum, and not much below the superior edge, a dark granular deposit was very distinctly seen to occupy the position of the large veinules and capillaries, which here mainly pursue a course parallel to the anterior epithelial surface. Under Hartnack's No. 11, à, Immersion, no crystalline forms could be detected in the deposit, nor could it be discovered that the granular particles were contained within the bodies of cells of the surrounding cell infiltration. In one or two of the papillæ, scarcely visible traces of these dark granules could be made out at points along the capillary loop. The granules forming the above deposits were variable in size and color, the latter ranging from dark brown to black by transmitted light, and the former not differing from that of the granules often found in silver staining. They were sufficiently numerous to mask the vessel wall where they occurred.

The next point to be determined was; whether this dark granular deposit so distinctly limited to the course of the deeper vessels of the gum, was some form of the blood pigment, or was silver in some shape. Of the coloring matters of the blood, hæmatin and melanin are granular and dark-brown or black. The others are crystalline, and may be excluded from further notice here. Hæmatin is soluble in either an aqueous or a spirituous solution of ammonia. Melanin is not at all or only slightly soluble in a strong solution of cyanide of potassium. Oxidized silver is readily soluble in the latter solution. In order to distinguish between these three substances, the following tests were applied to several sections of the small portion of gum:—

Several sections, in which could be well seen the dark, previously described deposit, were macerated for a considerable time in an aqueous solution of ammonia, rather strong. Upon subsequent examination under the microscope, the deposit appeared not to have decreased in density or to have changed at all. Other sections were macerated a few moments in a strong solution of cyanide of potassium, and were subsequently placed in the field of the microscope. From these sections every trace of the granular deposit had vanished. A fresh piece of the choroid coat of the human eye, after staining with silver, and exposure to light, was placed in an equally strong solution of cyanide of potassium, and the effect was observed under the microscope. The black silver lines rapidly disappeared, but the pigment in the hexagonal cells and in the stellate cells of the stroma of the choroid remained untouched. One other micro-chemical test was applied. Either the chromate or the bichromate of potassium gives, with nitrate of silver, a precipitate which is red by reflected light. Several sections showing clearly the granular deposit, were macerated a moment in a drop of nitric acid, rather strong. The nitric acid was then removed, and the sections were covered with a drop of a solution of bichromate of potassium, in which they were permitted to remain a few moments. They were subsequently well washed and mounted in glycerine. Viewed by reflected light, and under a low magnifying power, the formerly dark deposit now gave a distinctly red reflex when seen in sunlight. The bichromate had, as usual, stained the mass of the tissue yellow, and this color had the effect of slightly modifying the red so as to produce a yellowish-scarlet or an orange tinge of red. Microscopic examination by reflected light, of the previously-mentioned unstained section mounted in balsam, showed a whitish or grayish reflex from the position of the deposit, which, by transmitted light, appeared dark and granular. From the preceding examination I am, therefore, inclined to think that it is highly probable that the mass of the dark granular deposit above alluded to, consists neither of hæmatin nor of melanin, but that it is composed of silver.

At the time the above cases were examined, as I had never happened to see any allusion to such early staining of the gums in advance of general argyria, I fancied that the observation was an original one. On coming to examine the literature of the subject, however, I soon found that this was far from being the case. Dr. George B. Wood states (Treatise on Therapeutics, etc., 2d ed., 1860, vol. i. p. 401), "happily, before the skin begins to exhibit this hue, a similar dark discoloration usually appears upon the gums and tongue and in the fauces, and serves, if observed, to put the practitioner upon his guard." This statement appears to rest upon the authority of the original observer since, in the United States Dispensatory (thirteenth edition, 1870, p. 1049), Dr. Wood states again, "according to Dr. Branson, an indication of the approach of discoloration is furnished by the occurrence of a dark blue line on the edges of the gums, very similar to that produced by lead, but somewhat darker." I have been unable to find Dr. Branson's communication on this subject, nor do I find that his observation attracted lasting attention, since, with the two exceptions above given, I have not discovered any allusion to this important phenomenon occurring as a premonitory sign of argyria, in any of the numerous recent works on Therapeutics which I have consulted. I am glad, therefore, to have been led, by observing anew this blue silver line on the gums, to call attention to Dr. Branson's original observation. The practical importance of this sign is considerable. I believe that it enables us to administer nitrate of silver internally with safety so far as any danger of discoloration of the skin is concerned,

provided that the gums be examined every few days, and that the use of the remedy be suspended as soon as the characteristic blue line is observed.

Another point of importance is its close resemblance to Burton's blue line on the gums, due to the saturation of the system with lead. Indeed, in some of the cases I have recorded, it would have been impossible to determine, by inspection alone, whether the blue line was due to lead or not. It may be said, however, that in other instances the color of the silver line is darker and more decidedly blue, and also that there is a tendency for the discoloration to extend to a greater distance on to the gum from the line of junction with the teeth, and also for it to affect the mucous membrane of the lips near the junction with the gums. It will hereafter be necessary, however, in any case where a blue line is observed on the gums, to investigate the history of the case not only with regard to the action of lead, but also of silver.

The safeguard which I have above proposed, and which, I trust, will prove to be a constant and reliable phenomenon, becomes of greater importance when it is borne in mind that the discoloration of the surface is, as a rule, permanent. I have known several cases where it continued unchanged after the lapse of twenty years, and similar instances are not scarce in medical records. It is true that Orfila (Medical Times and Gazette, March, 1852, p. 279) found silver in the liver of animals, to which the nitrate had been given, six months after its administration; but found that in seven months it had disappeared from the tissues of animals similarly treated. And so, in certain cases, argyria has been stated to gradually diminish with

the progress of time. So, too, success has been claimed for various remedies in the treatment of argyria. Thus repeated blistering, alkaline baths, the prolonged use of cream of tartar and iodide of potassium, have all been recommended. Of these, iodide of potassium has most evidence in its favor, and is the one on which I should myself rely; but still in several instances it has been vigorously employed without success.

After the discontinuance of nitrate of silver in Case IV., iodide of potassium was directed with a view of favoring the absorption of the indurated cellular tissue surrounding the cæcum. This has given the opportunity of observing the influence of time, in connection with the use of the iodide, upon the blue line on the gums. At the present time, six months after discontinuance of the nitrate of silver, the line has perceptibly, though slightly, faded in intensity.

It is very possible that the difference in the persistence of the argyria, and in the results of treatment in different cases, may depend somewhat upon the amount of silver which has been absorbed and fixed in the tissues.

CASE

OF

MALIGNANT DISEASE OF RETRO-PERITONEAL AND MESENTERIC GLANDS, RIB, AND VERTEBRÆ; SECONDARY TUMORS WITHIN THE SPINAL CANAL, CAUSING PARAPLEGIA. WITH REMARKS.

By

JAMES H. HUTCHINSON, M.D.,

ONE OF THE ATTENDING PHYSICIANS TO THE PENNSYLVANIA HOSPITAL, ATTENDING PHYSICIAN TO THE CHILDREN'S HOSPITAL, ETC.

[Read April 4, 1877.]

The Fellows of the College may perhaps remember that at the meeting in May, 1875, I reported a case of Adenoid Disease, which was characterized by an unusually extensive distribution of lymphadenomatous tumors throughout the body, and in which paraplegia was caused by the compression of the spinal cord by one of these growths. To-night I wish to bring to their notice a second case of paraplegia resulting from the presence of tumors within the vertebral canal, and, although the nature of the constitutional affection under which the patient suffered was different, resembling the first case in the extent to which the disease was diffused.

I will read without further prelude the history of the case which I have drawn up from notes taken at the bedside by Dr. Morris J. Lewis.

S. M., æt. 50, a widow, and by birth an Irishwoman, first came under my observation during August, 1876, while I was temporarily in charge of the medical wards of the Pennsylvania Hospital. Upon being questioned in regard to her previous history, she said that, prior to the beginning of her present illness, she had always enjoyed good health, and that, as far as she knew, she inherited no predisposition to any form of disease. She admitted having been at one time rather intemperate, but denied that she had ever had any venereal disease; and indeed the only ground for suspecting the existence of a syphilitic taint, was the presence of cicatrices upon her right leg and thigh, which she herself attributed to an attack of phlebitis which had occurred during her last pregnancy, sixteen years before. When admitted to the Hospital, she was suffering from pain in the back and shoulder, apparently of rheumatic origin, which yielded readily to rest and simple treatment, but gave place at a later period, when she had passed from under my care, to symptoms, such as anorexia, constipation, and tympanites, indicating digestive derangement. These, too, seem to have disappeared under treatment, for she was discharged in about a month's time, if not entirely restored to health, feeling sufficiently strong to resume her former position as cook at one of the large hotels in this city. A return to work, however, soon brought on a recurrence of the pain in the back, accompanied by pain in the sides, which compelled her again to seek relief at the Hospital.

Upon her re-admission, on November 3, she was much emaciated, and complained bitterly of pain in her back and side. She was also suffering from constipation and anorexia, but the tympanites, which had formerly been the source of much distress to her, was no longer present. A careful examination revealed only well-marked pleuritic friction in the left infra-axillary region, together with some impairment of resonance on the same side of the chest. The urine was found to be cloudy, amber-colored, and acid. Its specific gravity was 1015. It contained urates in abundance, but no

albumen or sugar. Although the presence of pleurisy was distinctly recognized, she was placed upon good diet, quinia and cod-liver oil, her condition contraindicating all antiphlogistic measures. In spite of this treatment the patient did not gain strength, but, on the contrary, it became evident that she was daily growing weaker. In addition to the pain in her back, which was the cause of much suffering, especially upon movement—the patient often saying that she felt as if her back were breaking—she complained of shooting pains in the course of the sciatic and crural nerves. The lower dorsal vertebræ were also found to be tender upon pressure.

About a week after her admission, the patient directed our attention to a pulsation, seated midway between the ensiform cartilage and the umbilicus. Upon examination, a hard mass was found in this position, overlying the aorta and transmitting its pulsation. The tumor appeared to be deeply seated and firmly fixed in the abdomen, and was most distinctly felt just above the umbilicus. It could be traced, when the patient was in the recumbent position, from a point an inch and a half to the left of the median line, to one three-quarters of an inch to the right, and was painful when firm pressure was made over it. When she was lying upon her right side, a mass could be plainly felt on the left side of the abdomen, which careful palpation showed to be continuous with the one in the median line. No similar mass could be felt on the right side, and the inguinal glands, although distinctly felt, did not seem much if at all enlarged.

On the 26th of November, about three weeks after admission, the patient complained of a feeling of constriction around the waist, the pain in the back continuing without modification. After this, the lower extremities gradually lost the power of motion, until, on December 9, the paralysis became complete. The next day, retention of urine was added to the other symptoms. There was, however, no relaxation of the sphincter ani, the patient being still obstinately constipated.

The occurrence of paralysis of course led me to re-examine the case very carefully, when the tumor was found to have increased very considerably in size, reaching then on the left side as low down in the abdominal cavity as a line drawn from one anterior superior spinous process to the other. A hard immovable mass was also felt for the first time in the lower part of the right iliac region. In the course of the transverse colon, above the umbilicus, there were a number of nodular masses, suggesting at first the idea that there had been a rapid development of cancerous tumors in the peritoneum. A closer examination, however, showed that they were really scybala; a view of their nature which was fully confirmed by their disappearance under the action of a brisk purgative. There was also slight œdema of the legs, together with some prominence and enlargement of the abdominal veins. After the patient's admission, vomiting only occurred as a very infrequent symptom. The stools were several times inspected, but were never found to contain undigested fat.

Together with the loss of power in the lower extremities, there was a decided diminution, although not an entire loss, of sensation in the part of the body below the waist. All forms of sensation were not, however, equally affected, analgesia being present in a much more marked degree than anæsthesia. Thus the patient was able to say with a tolerable degree of accuracy which toe was touched, long after the prick of a pin had ceased to cause a painful sensation. Electro-muscular contractility and electro-muscular sensibility were at this time very much diminished in both legs, especially in the right one. Reflex action was almost entirely abolished. An examination of the blood under the microscope showed an excess of white corpuscles, as many as thirty-eight being present in a single field (400 diameters). Dr. J. G. Richardson, who was kind enough to repeat this examination, estimated the proportion of white to red corpuscles as one to seventy-five.

There is little to add to this history, the patient gradually sinking until death came to relieve her of her sufferings, on

December 27, about eight weeks after admission, and about three and a half weeks after the occurrence of paraplegia. During the patient's stay in the Hospital, her temperature rarely rose above the normal, until just before the close of life. It was, on the contrary, frequently subnormal.

The post-mortem examination was very carefully made by Dr. Morris Longstreth, the Pathologist of the Hospital, and to him I am indebted for the following very full notes:—

Autopsy, made eight hours after death: Rigor mortis well established; emaciation more marked on upper part of body than on lower; superficial veins more marked on upper than on lower extremities; no œdema; no general discoloration of surface; slight suggillations.

Thorax.—Left pleura contained some bloody serum. Adhesions of lung to diaphragm, and posteriorly; the lung was torn on removal; there was seen upon its surface a good deal of pretty firm lymph. Right pleura contained serum looking almost like pure blood; lung not adherent. No enlarged glands in anterior mediastinum.

Pericardium contained a small amount of bloody serum; heart's surface partially covered with fat; along its great vessels its adjacent tissue was stained bloody (imbibition). Large white patch on base of right ventricle, which contained a little black, fluid blood. Leading through orifice of pulmonary artery was a firm clot, whitish on one side, red on the other. Right auricle contained black, fluid blood, and a white clot. Left ventricle empty. Left auricle contained a small amount of black clot. Neither side of heart very rigid; the left cavities nearly closed (systole). Weight of heart, seven ounces Av.; flabby, easily torn, and appeared stained. Walls of ordinary thickness, valves and orifices presented normal conditions.

Upper and anterior part of lesser lobe of right lung pale; posterior part of lesser lobe dark, crepitant, moist; on section exuded bloody, frothy serum; surface of section not granular; on pressure, tissue broke down under the finger

more readily than is usual. Section of upper lobe seemed normal, but exceedingly pale. Left lung presented in general the same appearances on the surface as the right; on section the upper lobe less pale; the lower lobe presented the same condition as the opposite side.

After removal of heart and lungs, one or two enlarged glands were found white on section, but with central softening not marked. In right pleural cavity, close to vertebræ, and extending outwards about two inches, was a low, flat swelling, extending from fifth to ninth ribs, but most marked from seventh to ninth. At the left apex, occupying the length of the second rib, was a flat swelling, extending from the angle forwards to within one inch of the costal cartilage; this projected outwards into the axilla, and inwards, encroaching upon the pleural space, and fluctuated upon pressure. In the axilla, the vein rested in contact with the mass; in the pleural space, the serous membrane covering it seemed unchanged, although more injected than the adjacent membrane. The enlargement extended also laterally, occupying nearly completely the contiguous intercostal spaces, and the whole enlargement seemed to be bounded and inclosed by the distended periosteum of the rib, the continuity of which was destroyed.

Abdomen distended; gas escaped on section of abdominal walls. Liver reached three inches below ensiform cartilage, and one inch below border of ribs upon right side. Stomach lay below it, having an hour-glass contraction near pylorus. Omentum spread evenly over intestines, its vessels finely injected (venous); one large vessel at upper part contained a firm brownish clot of some extent in length. Fluid in abdominal cavity, bloody in character; no lymph or evidences of inflammation. Diaphragm reached in height to fourth interspace on right side.

Weight of spleen, five ounces Av.; small; border fissured; moderately firm; on section, pulp was soft, and fibrous tissue not prominent; on anterior border of spleen was a firm whitish nodule, size of hazel-nut; on section it was seen to occupy

whole of splenic tissue at that point; its outline was very distinct, and its surface showed streaking, as if traversed by vessels. Left kidney large, flabby, and irregular in outline; the capsule stripped off in removing the organ; surface very much mottled; at some points, dense, white specks, rising slightly above surface; spots of dense, red color were also seen; both of these were numerous. On section the appearances were normal. Weight five and a half ounces Av. In the connective tissue surrounding the right kidney were numerous white specks, much resembling tubercles; ureter and pelvis of kidney filled with urine; this kidney was larger than the left, and was also flabby; on its surface was seen one large white spot similar to those upon the left. These spots penetrated but very slightly into kidney-tissue; otherwise the two organs had the same appearance. Weight seven ounces Av.

There was a large mass occupying the mesentery; it was lobulated and nodular in character, and occupied nearly the whole of the tissue from the vertebræ to the intestine. The retro-peritoneal glands were also affected; they extended from in front of the bodies of the vertebræ, around on each side to the transverse process; above, they reached the diaphragm; below, the masses passed over the promontory of the sacrum, covering the whole anterior surface of that bone. The rectum was surrounded by these masses. Thus the basin of the pelvis was filled, crowding the uterus and ovaries forwards and upwards. The mass extended also to sides of pelvic basin, lying on and compressing sacral plexus of nerves, especially upon right side. A chain of enlarged glands extended from promontory of sacrum along brim of pelvis on right side into right iliac fossa; the last enlargement of this chain was near the anterior abdominal wall at the anterior superior spinous process of the ilium. Enlarged glands were also found in the line of the splenic artery, and in the lesser omentum, upwards to the transverse fissure of liver; in the adhesions of the stomach at its cardiac end, were similar, isolated masses.

Weight of liver, two pounds twelve and a half ounces Av.; several enlarged glands in transverse fissure; organ very pale and smooth; outline regular; mottled. On section, tissue had homogeneous appearance near surface, but in depth of both lobes was spongy, giving the organ an emphysematous feel and appearance. Near under surface, close to transverse fissure, was found a rounded mass about size of small walnut; this, on section, was found to contain whitish, thick fluid; its walls were thick and fibrous.

Supra-renal capsules were softer than ordinary, but otherwise appeared normal.

The internal organs of generation presented nothing abnormal. They were surrounded by, and adherent to, the mass found in the pelvis, above described.

A section through the mass occupying the mesentery showed isolated glands surrounded by adipose tissue. The centres of most of the glands were fluid in consistence. In some, the periphery (perhaps not glandular tissue but thickened surrounding tissue) was of a pinkish-white color, firm, and yielding little or no juice upon scraping. There was only one mass about the size of a walnut that presented no central softening. The whitish fluid from one or two of these glands was examined microscopically, shortly after the autopsy, and presented lymph-corpuscles and cells, with a nucleus and nucleolus, and somewhat fusiform in shape; some of these appeared rather as flattened cells, with fibres extending from or attached to their two poles. In this fluid were seen no large cells, nor other abnormal appearances.

Brain.—The vessels at the base of the brain presented nothing special, and the membranes, brain-tissue, and cavities were normal. The anterior extremity of the left middle lobe presented a depression about half the size of a hen's egg, but more shallow. This indentation corresponded to a prominence in the left middle fossa at the anterior part. Here the dura mater was smooth and covered a slightly fluctuating tumor. On raising the dura mater, it was found slightly thickened over the whole anterior part of the fossa, but not

very adherent. The tumor was found to be a mass having its origin in the bone as well as around it. On examining the external surface in the temporal fossa, there was found to be a similar tumor, like the growth from the bone within, and just in front of the zygomatic process of the temporal bone. Viewing the growth as a whole, it appeared in form similar to a small hen's egg, half being outside and the other half within the cranial cavity. The condition of the bone was similar to that of the rib already described, viz., its structure destroyed and its place occupied by a mass mostly fluid in consistence. There were found here, as in the ribs, masses of bony tissue, probably the compact portions rendered porous and friable. Surrounding the tumor-like growth in the left middle fossa, were numerous small, low, mammillated projections which extended especially towards the body of the sphenoid bone. The other portions of the skull were normal.

Spinal Cord.—Several of the vertebræ, especially in the dorsal and lumbar regions, were softened, so much so that it was possible to divide their bodies with a knife. On opening the cavity of the spine, there were found small masses within and adherent to the dura mater; these were irregularly distributed in its length, none occurring at the upper part, whilst in the lumbar and dorsal regions they were numerous and in places continuous. They were not of large size, yet in places pressed firmly on the cord; the pressure seemed most marked at the point where, in the cavity of the right pleura, the rib and vertebræ were found most affected, viz., at the position of the ninth dorsal vertebra. The pressure on the spinal cord seemed not wholly to be due to the size of the masses adherent to its dura mater, but also to a narrowing of the cavity of the spine by the changes in the bone itself. The consistence of the cord in the lower dorsal region was lessened, but above and below this part it gave the feeling of firmness. There were no masses found on the anterior surface of the cord, and none at all in the sacral

cavity, although the masses in the pelvis reached somewhat into the anterior sacral foramen.

The above wood-cut, from a drawing by Dr. F. C. Hand, represents the appearance of the spinal cord after the cavity of the arachnoid had been laid open.

The spinal dura mater was slightly adherent to the arachnoid in the lower dorsal region, but was free through the rest of its extent. The vessels of the pia mater were full, but there was no appearance of injection amounting to a morbid condition. On section of spinal cord at place of

lessened resistance in dorsal region, the surface welled up after the knife, but there was no diffluence; other portions were of normal resistance and wanted this phenomenon on section. No section examined presented any alteration of color or outline of either gray or white portion.

Portions of the tumors were sent to Dr. J. G. Richardson, Microscopist to the Hospital, who has sent me the following note:—

"On microscopical examination, I found that scrapings from the costal tumor and from a nodule removed from the spinal canal, showed large numbers of epithelial cells, caudate, triangular, and multangular in their contour, generally furnished with large nuclei. Thin sections cut after partial hardening in alcohol displayed similar cell elements, arranged in large alveoli of a rather scanty stroma. Although in section these growths have a marked resemblance to lymphadenomata, yet the diversity in size and shape of the cells, their occasional multinucleated character, and the relatively great magnitude as well as the irregular outline of these nuclei, lead me to rank these tumors as encephaloid carcinomata."

The condition in which the second left rib was found was a surprise not only to myself but also to all those who had seen the case with me during the patient's lifetime. Inasmuch as the left infra-clavicular space was repeatedly examined by percussion, shortly after the patient's admission into the ward, without causing her the slightest pain—which would hardly, it seems to me, have been the case if the disintegration of the bone had begun at that time—I am convinced that the rib must have become involved in the general disease after she came under my care. After these examinations had shown that, with the exception of the pleurisy at the left base, the lungs were healthy, I

thought it unnecessary to worry the patient longer in this way; there was, moreover, at no time any redness, swelling, or other symptom calculated to excite her attention or that of her physician. For the same reason, the tumor near the zygomatic process of the left temporal bone escaped detection during her lifetime, and even after death it was not discovered until the tumor within the skull was found to have penetrated the bone. It will not be without interest to add that there was no choking of the optic disks in this case, notwithstanding that the tumor was in the immediate neighborhood of the left eye.

The paralysis of the lower extremities appears to have come on rather slowly at first, the patient finding herself unable to walk some time before she lost the power of moving her legs in bed. Complete paraplegia came on, however, at the last, suddenly, and was quickly followed by retention of urine. The tumor could be felt reaching down so low in the abdominal cavity that it was thought at first that the paralysis was caused by pressure upon the sacral plexuses, which was indeed found to exist after death. A careful examination showed, however, that there was anæsthesia of the buttocks, and of the back as high as the waist, which, together with the paralysis of the bladder, could only be ascribed to a lesion of the spinal cord.

In the case which I have reported to-night, analgesia was much more marked than anæsthesia. In a condition in which it is obvious that all parts of the cord must suffer, and in most cases equally, it is difficult to understand how one form of sensation can be abolished, while another persists. But my observation is by no means isolated. In a case reported by

Sir William Gull (Guy's Hospital Reports, 3d Series, vol. ii.), he distinctly says, "The paralysis of motion in the lower extremities and sphincters became complete, but he [the patient] retained the power of distinguishing the seat and direction of superficial impressions on the skin, though no amount of pinching or pricking gave rise to pain." Rosenthal, in his *Klinik der Nervenkrankheiten*, reports at least one case in which a similar condition was observed, together with a loss of the power of distinguishing between different temperatures, and alludes to it as of frequent occurrence.[1]

A circumstance which excited a good deal of interest at the time of its discovery, was the large relative increase in the number of white blood-corpuscles. A careful examination by Dr. J. G. Richardson showed that they were only four times as numerous as in health, but, when the blood was first placed under the microscope, the appearances really seemed to indicate the presence of leucocythæmia. In fact, so great was their apparent number that I abandoned for a time the belief that the tumors were malignant, and was inclined to regard the case as one of adenoid disease. In cases of cancer of the abdomen it is, however, customary to find a large increase of white corpuscles. Indeed, Dr. Moxon (British Medical Journal, Dec. 16, 1876, p. 793) says that he has seen one white for every twenty red corpuscles in such a case, and that he has used this fact as a means

[1] According to Schiff, lesions of the gray substance of the cord produce analgesia, while, on the other hand, anæsthesia is a constant accompaniment of diseases of the posterior columns. This is not admitted by Brown-Séquard and Vulpian, who claim that the different varieties of sensation are conveyed to the brain by a system of conductors in the gray matter alone.

of diagnosis, in a case where it seemed at first doubtful whether he was dealing with idiopathic anæmia or with gastric cancer. The increase of white corpuscles may, however, after all be dependent, I think, upon the fact that with the abdominal cancer there is as a necessary consequence more or less enlargement of the neighboring lymphatic glands.

Tumors growing in the cord, or from its membranes, are, according to Sir William Gull (Guy's Hospital Reports, 3d Series, vol. ii. p. 143), among the more rare causes of paraplegia. If this statement be modified so as to read tumors within the spinal canal, and in this way be made to include growths from the vertebræ, it can no longer be accepted as strictly correct; for, in addition to the two cases which have occurred in my own practice, I have been able to collect upwards of fifty others, and a reference to the Bibliography, which I have appended to this paper, will show that this number might be readily much increased.

In the history of the case of adenoid disease which I reported to the College two years ago, I mentioned that, until it came under my observation, the cause of the paraplegia was believed to be simple myelitis. Unquestionably myelitis was present in this case, for the spinal cord presented the appearances usually found in that disease; and the notes of the case which I have just reported show that in it the cord was similarly affected. But, as myelitis is a rare disease, it will readily, I think, be admitted that it would not have occurred had it not been for the irritation occasioned by the spinal tumors. Whether or not the paraplegia, pain in the back and legs, and the other symptoms present

in these two cases, would have occurred if the inflammatory complication had not arisen, is a question worthy, I think, of a good deal of consideration. In other words, Is mere pressure upon the spinal cord insufficient to give rise to paralysis, and may intra-vertebral tumors, as is frequently the case with intra-cranial growths, remain latent until they have excited organic changes in the surrounding nervous tissues? From a careful study of the cases which I have collected, I cannot but conclude that, except in a few instances, these questions must be answered in the affirmative. Paraplegia does, it is true, frequently occur as the result of the great pressure to which the cord is suddenly subjected in cases of fracture or dislocation of the vertebræ, or it may follow closely upon the rupture of an aneurism of the descending aorta into the spinal canal, as in the celebrated case reported by Laennec (Traité d'Auscultation, Paris, 1828, p. 623), but the compression by a tumor is more gradual, and the cord has probably the same power that the brain has to accommodate itself to a certain amount of pressure. When, however, a certain degree is reached, the cord is no longer able to resist the irritation, and myelitis is set up; and the resulting paraplegia is attributable rather to this complication than to the original disease.

This view is sustained (1) by the fact that the pain in the back, a very characteristic symptom of tumor, often antedates the paraplegia by quite a long interval. This was notably true of the cases reported by Sir Wm. Gull (Guy's Hospital Reports, 3d Series, vol. ii.), in one of which the pain preceded the paralysis by from three to four years. There was no disease of the

vertebræ in this case, so that the pain cannot fairly be ascribed to erosion of these bones, as it might be in the case which I have reported this evening. (2) When the tumor occupies the cervical portion of the spinal canal, brachial diplegia, from pressure upon the roots of the spinal nerves, may occur some time before the lower extremities are involved in the paralysis. (3) In very few of the reports of the cases which I have already referred to, is it distinctly said that there was no myelitis. In not more than six out of thirty-five cases, which have been studied with special reference to this point, are we justified, from the account of the post-mortem appearances, in concluding that it was absent; in four, the condition of the cord is not noted; while in all the others it is either expressly stated that myelitis was actually present, or, as more frequently happens, the cord is said to have been softened, or almost diffluent, and to have welled up upon section.

Moreover the view above given is the one taught now by both Charcot and Rosenthal. The former of these distinguished observers has recently expressed the opinion (L'Abeille Médicale, Jan. 18, 1875), that the paralysis in Pott's Disease, which it is customary to attribute to pressure upon the cord by the displaced vertebræ, is really due to the presence of myelitis as a complication. The latter holds (Klinik der Nervenkrankheiten, p. 313), that the seat and extent of the consequent central lesion have more influence in determining the symptoms than the nature of the pressure cause.

Pain[1] would appear to be one of the earliest, as well

[1] In seventeen cases in which there was no disease of the vertebræ, the tumor being seated wholly within the spinal canal, pain of some form or

as one of the most prominent, symptoms of compression of the cord by tumors. It is generally seated in the back, and more or less correctly indicates the position of the disease. Sometimes, along with this pain or independently of it, the patients complain of pain in the shoulders, arms, legs, or some other parts of the body, in these cases unquestionably indicating the existence of pressure upon nerve roots. The pain may in some instances be intermittent, as in a case reported by Mr. Aston Key (Trans. Path. Soc. Lond., vol. ii. p. 26), in which pain in the left shoulder and in the posterior thoracic region was complained of nearly two years before the death of the patient, which followed almost immediately upon the occurrence of paraplegia. The pain, however, yielded at first to rest and the other remedies employed, but reappeared with its former intensity about a year subsequently. Relief was again obtained, but only for a time. Together with the pain in the limbs, there are very apt to be associated abnormal sensations, as numbness or tingling. The sensation of constriction around the body is generally a later symptom, and is probably rather dependent upon the intercurrent myelitis than upon the pressure of the tumor. Pain was a very marked symptom in the case which I have reported this evening, the patient often saying that she felt as if her back were breaking. It will be recollected also that the pain was relieved for a time by rest, to reappear when work was resumed.

other preceded the other symptoms, and in most instances by a decided interval. Paraplegia, on the contrary, was a late symptom, and its onset was usually accompanied by other symptoms which are generally referred to myelitis.

Muscular twitchings are also frequently observed, and when they cannot be readily explained should always excite suspicion of serious disease. They are probably a result of irritation rather than of inflammation of the spinal cord, and in point of fact appear to be oftenest noted before the occurrence of paraplegia. This is also true of swelling of the joints, when it occurs (for this does not seem to be a frequent symptom), and of increased reflex action.

Symptoms belonging properly to myelitis are, as I have attempted to show, paraplegia, impairment or loss of the different varieties of sensation (anæsthesia and analgesia), loss of power over the bladder and sphincter ani, permanent contraction of the limbs, and bedsores.

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THE STATE OF MEDICINE IN CHINA.

By

ROBERT P. HARRIS, M.D.

[Read May 2, 1877.]

Among the numerous articles in the Chinese section of the late Centennial Exhibition, there was nothing more curious, to the eye of a practitioner of medicine, than the collection of specimens of Materia Medica, embracing several hundred articles, used by the native Chinese doctor in the treatment of disease.

I shall not attempt to enumerate the various articles taken from the animal, vegetable, and mineral kingdoms, many of them objectionable, some highly disgusting, and a very few known to us, which enter into the Chinese Materia Medica, but will refer the curious to the catalogue which I have procured for the library of the College of Physicians from the Chinese Commissioner, in which will be found the value and uses of the several medicines, and from which may be learned how little of Chinese therapeutics is based upon a course of reasoning from cause to effect.

The fact that ginseng is still regarded in China, after centuries of trial, as a drug of marvellous healing, strengthening, and rejuvenating powers; and that liquorice root is esteemed only second to it in medicinal effects, shows how little connection there is between the real and conjectural value of Chinese medicines. The scarcity of a remedy, or the difficulty of obtain-

ing it, appears to greatly enlarge its value to the Chinese mind. When American ginseng (*Aralea quinquefolia*) was first exported to Pekin, it sold for its weight in gold; but, being abundantly supplied, it gradually fell in estimation until now a pound is valued at about as many cents as an ounce formerly brought in dollars. The native varieties of Aralea, which are found wild, and only in small quantities, in Chinese Tartary, have, on the contrary, advanced in estimation, so that the price in Canton is reported at $26 per ounce. Glue is also considered medicinal, but is of very little account unless the variety is prepared from materials difficult to procure, such as that from tiger bones, which is sold at $11 per pound, etc.

Thus it will be seen, from articles of known value, how little faith is to be placed upon the Chinese estimate of any medicinal plant or product, although possibly having some remedial virtue. The main basis of Chinese therapeutics appears to rest upon mystical ideas; and the cure of disease is divided between the *exorcist*, the *diviner*, the *charlatan*, and the *doctor*, all of whom are believed by the native to deal in mysteries, and to derive the power through special divinities whose name is legion. In fact, the complicated religious belief of the Chinaman appears to make it almost impossible for him to reason from cause to effect in direct medication, even in the management of the most simple maladies.

Conjunctivitis, entropium, pterygium, sometimes double in both eyes, and cataract, are common maladies in China, especially the first two; and blindness from defective medical treatment in ophthalmia, very abundant; still the native physician does not appear

to have the least atom of common sense in treating even a simple inflammation of the conjunctiva. Imagine the sensation felt, and the result produced, by introducing a little pulverized and levigated *stalactite*, *fossil crab*, or what they call *medicine stone*, into an inflamed and highly sensitive eye; these being some of the remedies in use.

As the "*Outside Barbarian World*" was but a few centuries ago almost on a medical par with the inhabitants of the "*Flowery Kingdom*," we might reasonably hope for the same degree of enlightenment in a proportionate period, if it were not for the very strong opposition of the better classes of the Chinese to everything coming from foreigners, whether in the form of customs, change in dress, religion, or improvement in the medical or surgical treatment of disease. We say better classes, because the poor, who are most likely to suffer from bad or defective medicine and surgery, have been very readily induced to submit their cases to the care of English and American practitioners offering their services gratuitously. This prejudice applies not only to foreigners and foreign physicians, but also with almost equal force to those of their own countrymen educated in and practising foreign medicine. As there is no Chinese surgery of an operative kind, the work of the knife is gradually effecting a change, and preparing the way for the introduction of a rational system of medicine which must ultimately culminate in the opening of scientific medical schools under Chinese professors, and possibly even fostered by the government. There are great obstacles to be overcome, but the work of the last

forty years has effected such changes that a full fruition would appear to be only a question of time.

Physicians and apothecaries abound in all the large Chinese cities, and, strange as it may appear to us, they have such as are recognized as *regular*, and others that are regarded as *quacks*. The first requisite for a Chinese doctor, is hereditary descent; and this is a certain guarantee of success in gaining practice if a long line of ancestry shall have transmitted the virtues of the *medical succession*. The sign of a doctor often informs the stranger, not only of ancestral qualification, but states his several specialties as handed down, and claims for him the possession of valuable formulæ.

The Chinese doctor, like his brethren of India and Japan with some few exceptions, has not the remotest conception of the internal anatomy of his patient; and his expressed idea is that "it is not necessary to know what is in a man to know how to treat his diseases." Those who saw the old Japanese manikin exhibited in the collection of models, instruments, etc., before the members of the American Medical Association in this hall in 1872, will remember how remote the conjectural idea was from the real anatomy of the abdominal viscera.

As the dissection of the dead is not a part of his education, the Chinese physician has not the remotest idea of the arrangement of even the larger viscera. He places the heart in the centre, and believes that it is surrounded with six organs, three of which are connected with the pulse of one wrist, and three with the other. To feel the pulse *with ability*, is considered the important inherited faculty of the practitioner.

When called to a case of disease, he feels the pulse in each wrist *with three fingers*, asks the age of the patient, inquires as to the symptoms, assumes a grave importance, and then writes out a prescription, which in general calls for a number of ingredients to be made into a decoction; or a number of pills, the quantity to be taken being a marvel to any but a Chinaman; or it may be a bolus, or other form. This feeling of the pulse in both arms, and for a long time, in ignorance of the synchronism of the radial beats, is mentioned also of the Hindoo and Japanese physicians, who, with a few exceptions, have likewise no knowledge of the circulation of the blood.

As there are no native schools of medicine in China, an aspirant for medical honor, if he has not the advantage of being the son of a physician, simply engages himself to one, that he may witness his practice, and learn as many of his secrets as he may be willing to part with. The pupil is taught to believe that the power to heal disease lies in the assumption that the human constitution is composed of five elements in different proportions, all acting in harmony during health, and each presided over by a different deity. These elements are *earth*, *fire*, *gold*, *water*, and *wood*. As long as the proper proportion of each of these is maintained, the health remains good, but any ascendency of either one deranges the system, giving rise to diseased phenomena, to be recognized by the physician, and counteracted by proper antidotes, or regulators. An example of this kind of practice is given in the history of the English embassy to China, under Sir George Staunton, in 1793. One of the embassador's suite became affected with dysentery, stopped at

a Chinese inn, and was persuaded to consult a native physician, who, attributing his disease *to a predominance of cold humors*, prescribed strong doses of pepper, cardamom, and ginger, in hot distilled spirit, by which the disease was so much increased that the patient made a very narrow escape for his life.

As the cure of disease is very largely credited to the influence of the special deity having charge over the deranged element involved in any given case, large sums of money are annually expended by restored patients in processions and feasts in honor of the said gods, a portion of the year being specially devoted to these religious ceremonials.

Chinese physicians frequently combine necromancy and fortune-telling with the practice of medicine. They have one curious custom that prevails all over the Empire, which is to deposit in the street or road, opposite or near the house of the patient, the residuum of any prepared infusion or decoction prescribed for him, under a superstitious belief that there is at night a roaming divinity on patrol to take care of all diseased persons. This "*Spirit of the Celestial Cure*" (Tien-i-star) is believed to make his rounds on horseback, and the object of the deposit is to induce the charger to take a sniff at it in passing, by which it is held that the rider is influenced to look with favor upon the case of the patient.

A prevalent error with us is the belief that the Chinese pay their physician while in health at a certain rate, and stop payment during sickness. A bargain based upon a prospective cure, with no charge except for the medicine in the event of a failure, is frequently made, but this belongs more to the pro-

vince of quackery than to regular practice. The general fee for medical attendance varies from sixty to two hundred and forty cash, or from four to sixteen cents per visit.

Native surgery has no real existence, as the knife is rarely if ever employed. A compound comminuted fracture ends usually in gangrene, and frequently in death, as amputations are not performed even for such injuries. Abscesses are very rarely evacuated, and dislocations remain unrestored, being simply dressed with some secret vulnerary ointment.

We come now to a more interesting branch of our subject, viz., the efforts that have been made to introduce legitimate, or "western medicine" into China. Although some slight progress was made prior to that time, the first important step may be said to have been the opening of the Ophthalmic Hospital of Canton, under Dr. Peter Parker, an American, in 1835. This specialty was chosen for two reasons; the eye of the Mongolian is particularly subject to disease, and the treatment of ophthalmic cases involves no risk to life. It being the rule to make the physician accountable for any fatal result in cases where the knife was used, a written guarantee of immunity was obtained from each patient before an operation, as it was important to run no risks until an inspired and enlightened confidence should remove this personal accountability. By this plan, Dr. Parker gained the confidence of the natives, and was by slow degrees enabled to extend his surgical practice, until he could perform the capital operations without any fear to himself in case of a fatal result. From this beginning grew the present General Mission Hospital of Canton, with its

enormous out-door practice, and its branch dispensaries in which, collectively, as many as thirty thousand patients were treated and prescribed for in a single year, before it had been thirty years in operation.

Fractures and dislocations are very rare among the Chinese, who from carefulness, freedom from intoxication, and the absence of heavy machinery, are seldom injured in this way. Gunshot wounds inflicted by river pirates and in clan-fights, are much more common, and patients frequently carry balls for years, until they are finally removed at some foreign hospital or dispensary.

The practice of obstetrics is entirely in the hands of midwives, as it is considered indelicate for a man to attend a pregnant woman, or to officiate at her delivery. Foreign physicians, and properly educated natives, have in latter years been sometimes called in to use the forceps as a last resort, in cases where the midwives have utterly failed after a delay of from two to four days. Embryotomy was performed in 1860, for the removal of a putrid fœtus, by Drs. John G. Kerr and F. Wong, of Canton, this, as far as known, having been the first time it was ever done in China. This Chinese, Dr. Wong, is a graduate of the University of Edinburgh, and has been in Canton since 1857. He is a valuable assistant at the hospital, performs many important operations, and has a good private practice notwithstanding the prejudice that attaches to his foreign medical education. He is also an important teacher of medicine to the class of native students attending the hospital.

Insanity is said to be rarely met with in China; but this is believed to be due rather to its management than

to its rarity, although one fruitful source (alcoholic intoxication) being almost exceptional, the disease is no doubt less prevalent than with us. Mild cases are left at liberty; troublesome patients are shut up, and often chained at home, where they not infrequently commit suicide; and dangerous subjects are sometimes quietly put to death by their relatives. There has never been a lunatic asylum in China, and never will be until introduced by foreigners.

The Chinese claim to have inoculated for the small-pox as early as the tenth century. Their method one would suppose might produce a general eruption, as the virus is introduced into the nose, in the form of powder placed in cotton. The person to be inoculated is first prepared by a course of medicine and dieting, and a lucky day is chosen for the operation.

Vaccination was introduced into China about 1805, by Dr. Alexander Pearson, physician to the East India Company, who instructed a Chinaman of the Nan-Hoi district, named Yau-Ho-Chun, how to perform it. Yau made a special business of vaccination up to the time of his death in 1850, and his son became his successor. He published a book of one hundred pages upon vaccination in 1818, and several Chinamen are said to have made fortunes by becoming professional vaccinators. From this early beginning under Dr. Pearson, the practice of vaccination has advanced to most of the eighteen provinces of China, but is still very limited in its application, owing to obstacles resulting from superstition and prejudice, one of the great difficulties being the impossibility of obtaining a continuous supply of *good*

virus. The natives have a superstitious fear of having the vesicle opened; are very careless in, or opposed to, preserving the crusts; and do not like to have the operation performed in the warm season. Hence native vaccinators, to secure a continual supply of virus, have been in the habit of bribing the lowest poor to submit their children to vaccination and furnish them with the resulting lymph, a very objectionable practice, owing to the prevalence of syphilis and leprosy. To obviate these evils, the mission hospitals and dispensaries are in the practice of vaccinating the poor on certain days, and securing their return for inspection, and obtaining the virus when unobjectionable, by a small pledge deposit, to be refunded to them, a plan not uncommonly adopted in our own dispensaries. They also have the means of obtaining fresh virus by reciprocal exchange with each other, or from physicians in India. As smallpox is almost an annual scourge in China, the importance of vaccination cannot be overestimated.

Prior to the opening of the Ophthalmic Hospital of Canton, in 1835, by Dr. Parker, there were established a Macao Dispensary in 1820, an eye infirmary at the same place in 1827, and the Dispensary of Canton in 1828. Dr. Parker also established an ophthalmic hospital at Macao in 1838, and was the first to open a general surgical hospital and to teach medicine to the Chinese. His first pupil was Dr. Kwan Ato, who died in June, 1874, at the age of fifty-six. He was a valuable assistant surgeon, and did great credit to his preceptor. He had an extensive practice among the higher classes, and did much to introduce rational medicine into favorable notice. His uncle, Lamqua,

was an artist, and painted a number of oil pictures for Dr. Parker, representing patients prior to operation, from whom he removed large tumors. These pictures were shown and explained by the Doctor at the surgical clinic of the University of Pennsylvania, during the professorship of Dr. Gibson. When the rebels besieged Canton, Dr. Kwan Ato offered his services as a surgeon to the Empire, was accepted, and for his skill was honored by the Emperor with the title of Mandarin of the fifth rank.

From the opening of the Mission Hospital of Canton, down to the present time, it has been in a small and imperfect way more or less of a medical school; and now gives a promise of becoming a college for the education of the Chinese in all the branches of medicine. Besides Drs. Kwan Ato and Wong, already referred to, we find honorable mention in the reports of the hospital, of Drs. So-To-Ming, U. A-Chung, and others, with special reference to their skill in operations for stone, cataract, necrosis, removal of tumors, etc.

The want of medical books in the Chinese language, and the existence of an almost unconquerable prejudice against human dissection, or autopsies, has long been felt; but the former has been in large measure provided for, as text-books have been published on all the branches taught in our schools.

Anatomy has been demonstrated by the use of models and plates, and by the dissection of dogs; but recently an advance has been made in the occasional autopsy of a patient dying without friends; the dissection of a child, young subjects being less cared for by the Chinese than adults; and the hurried

examination of an adult arm or leg before the class. These have been openly done, to test the progress made in overcoming the prejudices of the natives, and to pave the way for future advances. As the Chinese worship their deceased ancestors, they have consequently a sort of reverence for a cadaver, and the use of one for scientific study is very abhorrent to their feelings: but this antipathy will in all probability give way, as many others have, in the progress of medical enlightenment.

The chief contributors to Chinese medical literature have been the late Dr. Richard Hobson and Dr. John G. Kerr, recently returned to our country after an absence of twenty-two years.

To Dr. Hobson is due the credit of having prepared the first medical work, An Outline of Anatomy and Physiology, published in Shanghae in 1850. His other works were on Physiology, 1851; First Lines of Practical Surgery in the West, 1857; A Treatise on Midwifery and Diseases of Children, 1858; and Practice of Medicine and Materia Medica, in the same year.

Dr. Kerr published a paper on Vaccination in 1859; Fever and Hernia, 1859; Materia Medica, 1871; Bandaging, 1872; Symptomatology, 1873; Skin Diseases, 1874; and Chemistry—the last, an octavo of 597 pages, in four fasciculi. The first two appeared in 1871, and one hundred copies were ordered for the Imperial College of Peking, and fifty by a bookseller in Yedo, Japan, where the educated natives understand the Chinese printed character.

Dr. Dudgeon, of the London Mission Hospital of Peking, published a series of Miscellaneous Essays

on Western Medicine, in 1875. Dr. F. P. Smith, of the Hankow Hospital, a work on Hygienics, in 1867; and Dr. Manson, of Amoy, a large work on Diseases of the Testes, in 1874.

A collection of these works, together with numerous, illustrated, hospital reports, and some medical pamphlets, were placed upon exhibition in the Chinese section of the Centennial. When it is remembered that it requires on an average a period of six years, under a teacher living constantly in the house of the pupil, for an American to obtain even a very imperfect knowledge of the Chinese language, the labor of preparing these medical works will be appreciated.

Mission Hospitals have been established at Taiwan, in the Island of Formosa; at Canton, where the ground and buildings belong to the Society; at Swatow, Amoy, Fooehau, Ningpo, Shanghae, and Peking; and in connection with these, or located at a number of inferior stations, dispensaries, the out-practice of which is in some instances immense, averaging over a thousand cases per month.

The Province of Kwang-Tung, in which Canton is located, abounds in cases of stone in the bladder, urethra, and prepuce, and furnishes a large number of subjects for the operations of lithotomy and lithotrity every year, amounting in 1875 to 31 of the former, and 20 of the latter; and for the whole period since the founding of the hospital to 332 of lithotomy, with 26 deaths, and 87 of lithotrity, with 8 deaths. As these cases are generally of long standing, the calculi are on the average unusually large. Tumors of very large size are also abundant, as the native

physicians do not remove them, and they are permitted to grow until their bulk and unsightliness compel their extirpation at the hands of a foreigner.

At Ningpo the chief diseases treated are intermittent and remittent fevers, rheumatism, pneumonia, ulcers, ophthalmia, cataract, and scabies. Victims of opium-smoking frequently apply for treatment, with a view to reformation, and some become broken of the habit, although the temptations to relapse are very great where the vice is so common. As the habit is not a social one, and there is no treating on the part of inviting companions, as in spirit-drinking, the reformation should be less difficult than that from the habit of drinking to intoxication is with us. Although very difficult to estimate, it has been thought by observers, claiming the requisite experience, that not more than from one to three in a hundred of the inhabitants of China smoke opium, and that these are mainly residents of cities and the larger towns. As with our drunkards, the cost to a habitual smoker is often half of all that he can make.

Among children, infantile convulsions, scrofula, and hereditary syphilis, are frequently met with. The uses of quinine in autumnal fevers, and of the truss in hernia, are becoming known to the Chinese through hospital and dispensary cases, and the dissemination of pamphlets. Surgical instruments are being made by native cutlers after European and American models, but the workmanship is inferior to that of the Japanese.

Native physicians and their sons are commencing to appreciate the superiority of a proper medical education, and are entering the Canton Hospital as stu-

dents. Thus the work of the gradual enlightenment of the Chinese mind upon the question of the relative merits of the native practice, and of the scientific methods of treating diseases and injuries as taught and employed by foreigners, steadily advances. Much of the prejudice felt by the Chinese could be done away with if the prescriptions given them by foreigners could be put up at their own apothecary shops; and, this being recognized, they are directed to them when practicable; but as yet the Chinese druggists pay little attention to keeping foreign articles such as we prescribe, and when they do professedly, the preparations, such as sulphate of quinine, etc., are not reliable. No doubt this will in time, however, be met by the education of natives in practical pharmacy.

Dr. Parker has lived to see a wonderful growth in the reputation of the Canton Hospital, since the time when a few suffering eye patients came almost by stealth to consult him at his office. The in-door cases are now about 1000 per annum, and the out-door somewhere about 18,000. The Hospital Society has also under its care four Dispensaries, and the whole number of patients prescribed for in all the institutions amounts to nearly or quite 35,000, as it reached 33,317 in 1875.

Dr. Happer, another medical pioneer, graduated in the University of Pennsylvania in the class of 1844, and arrived out at Canton the same year, where he has remained almost uninterruptedly ever since. The Canton Hospital has been supported mainly by the foreign merchants resident in China, but in latter years has been largely aided by Chinese government officials, merchants, and compradores.

Clinical reports, some of them of very remarkable cases, occasionally appear in European medical journals; such as those of Dr. Edward Henderson, of Shanghae, given in the January and February numbers of the Edinburgh Medical Journal for this current year. One of his cases would never have occurred in any country where operative surgery is legitimately practised. A young Chinaman, twenty years of age, became affected with urethral calculi at the age of four, and permitted them to multiply until his glans and prepuce were dilated into a calcareously lined cyst of twelve inches' circumference, containing thousands of small calculi, and weighing, after removal behind the corona, a pound and a half.

Calculi are frequently found in the urethra of the Cantonese, located near the end of the canal, and as many as 116 have been removed from the cavity of a constricted prepuce in a man of 72. In the northern regions of China vesical calculus is rarely met with as compared with the southern.

The peculiar form of the palpebral opening in the eye of a Mongolian, appears to render him especially liable to entropium as a result of a chronic conjunctivitis, and vast numbers present themselves for treatment. This character of the eye also materially interferes with the operation of extracting the lens for cataract, especially by the superior incision, and the inferior one is generally resorted to, although the other is preferred where there is competent assistance at hand. Operations for cataract are remarkably successful in Chinese subjects, and the peculiar, innate, manual dexterity of the natives appears to fit them to become skilful operators.

The remarkable prevalence of conjunctival inflammation among the rice-eating nations of the world, has given rise to an impression that this grain has an influence in producing it; but a careful investigation will determine the existence of a variety of causes in different localities, not connected at all with this diet. The purulent conjunctivitis of Egypt is believed to be due to uncleanly habits, inviting the attacks of flies, which thus transfer decomposing matter, obtained from dead animals or parts in a diseased condition, to the eyes—mostly of children. Not only is the eye of the Chinaman peculiarly susceptible to inflammation, but this is produced by a variety of influences. The cold and dust-storms of the north, the effects of heat, sunlight, bad ventilation, and exposure, especially in the water-boats, at the south, and of the over-straining of the eye in delicate handicraft, all act as exciting causes; and, when once produced, the inflammation is apt to become chronic, from pernicious or the want of proper medical treatment in the acute stage.

No one in China has had so much influence in overcoming, at least apparently, the antipathy of the natives for foreigners as the operative surgeon. There is no question that they at heart hate us, and will often be heard, by those understanding them, to apply the epithets, "*foreign devils*," "*outside barbarians*," etc., when talking among themselves in regard to us. But the surgeon appeals to their sense of gratitude by favors conferred, in restoring the blind to sight, removing diseased growths, etc., and thus secures not only their kindly feelings, but in some cases immunity from attack, and safety to life and property. He is

also enabled to obtain the *entrée* into the houses of the nobility, and thus secure the protection of those high in authority, and their co-operation in carrying on the work of medical reform.

Among the surgical patients applying for aid are great numbers whose cases belong to the domain of minor surgery, and who are readily relieved by the puncture of an abscess, or performance of some trifling operation; but, influenced by the success in these cases, are many other patients upon whom the surgeon is expected to exercise his skill, and is given full liberty to perform whatever operation he may think advisable, no matter how severe or hazardous it may be. Confidence begotten of success has done wonders for surgery in China, and very fortunately the habits and temperament of the natives greatly favor them in their recovery. Pyæmia is a very rare sequel, although such a drawback to success with us, where its occurrence is evidently on the increase. We believe that both pyæmia and gangrene are greatly due to the condition of the tissues and organs brought about by intemperance, and that the absence of organic changes in children and the Chinese, is largely due to freedom from the use of intoxicants, which will account for the rarity of pyæmia in both. In this respect the Japanese are an inferior race, being addicted to intemperance, and having very little recuperative power when operated upon.

Opium smokers are not good subjects for serious operations, as their systems are exhausted from the effects of the poison; those not addicted to the habit, or but slightly affected by it, generally do well.

As one of the methods of treating hemorrhoids

adopted by the native physicians is to apply escharotics (probably pulverized sulphate of copper, which is one of their remedies), patients present themselves with cicatricial strictures of the anus; in some cases. with the rectum almost entirely closed up. For this condition there does not appear to have been found any permanent relief.

Removal of the upper jaw has been several times performed at Canton; and a large aneurism of the carotid artery was successfully treated by digital compression, kept up by relays of the Chinese medical students. The method was very painful at first, and could not be long sustained, but in time the compression became much less severe, and was continued a number of hours uninterruptedly except for the momentary change of students. Treatment in this form lasted at intervals twenty days, and the patient was under observation for eighty-two.

Elephantiasis prevails in its most frightful forms among the Chinese, attacking in one subject the face, and particularly the mouth; in another, the upper extremity, enormously enlarging the hand; in a third, the scrotum; and in a fourth, the lower extremity, as in the Barbadoes leg. Operations have in some instances been performed, as upon the face, but not with any permanent benefit, as the hypertrophic enlargement is renewed.

The Chinese evince an excellent capacity for acquiring medical knowlege, and several have become very skilful in their surgical operations. At one period the Canton Hospital was conducted entirely by them for nine months, during which time the operations performed exceeded the average usual for the

same number of months, and at all times they now do a large share of the hospital work.

The great central organization, known as the "Medical Missionary Society," which was inaugurated at Canton, February 21, 1838, under Dr. Thomas R. Colledge, as its first president, and with a full corps of officers and directors, appears to have been well aware at its beginning of the importance of educating the Chinese in rational medicine, and evidently had it then in view to found a school for the training of native physicians and surgeons, as will be seen by reference to an address drawn up by Drs. Colledge and Parker and Mr. E. C. Bridgman, in April, 1838. To these three also is due the credit of taking steps to form this Society in December, 1835. Dr. Colledge was a surgeon in the British service, stationed at Macao, and divides with Dr. Parker the credit of having established the Mission Hospital of Canton, although its reputation with the natives was chiefly the result of the surgical skill of Dr. Parker, who very fortunately possessed the physique to endure the vast amount of work that he was called upon to perform. Some of the Fellows of this College still living, have the satisfaction of feeling that they aided in providing a fund with which this hospital purchased for its use several cases of much needed surgical instruments, that in their day did very important service.

Much credit is due to the Medical Missionary Society in China, for the persevering activity and faith with which it has labored to accomplish the ends for which the organization was founded thirty-nine years

ago ; and we congratulate its members upon the success they have met with in their endeavors to introduce "*western medicine*," and make it popular with the natives, both as patients and practitioners. It is certainly a matter of much professional interest to us to know that a revolution in medical ideas is being slowly effected even in China.

REMARKS

ON

RELAPSES IN TYPHOID FEVER.

By

J. M. DA COSTA, M.D.,

PROFESSOR OF THE PRINCIPLES AND PRACTICE OF MEDICINE IN THE JEFFERSON MEDICAL COLLEGE.

[Read June 6, 1877.]

During the epidemic of typhoid fever which prevailed among us last year, I met with many instances of relapse. This led me to investigate the subject anew; and in comparing the phenomena observed with those encountered previously, I have arrived at some general conclusions which I beg leave to lay before the College to-night. In so doing I shall not in detail record any large number of cases, partly because their similarity would make this wearisome, partly because some of them were seen but a few times with other physicians, and the salient features alone are impressed on me. But all have been made use of to arrive at the deductions about to be presented.

First let me say exactly what I understand by a relapse. I mean a return of the symptoms of the malady in all directions: return of diarrhœa, of eruption, of fever-phenomena, of delirium, of pulmonary congestions. I do not simply mean those slight and mostly transitory accessions of trouble which we some-

times see in consequence of bad nursing, as of the patient being needlessly disturbed, or allowed to take improper food. I mean, therefore, a fresh outbreak which runs a definite course, and is often protracted, and may be dangerous.

It is only of comparatively late years that the occurrence of true relapses in typhoid fever has been admitted, and even now the fact is denied by some; while accurate observations on the mode and cause of the return of the affection are still greatly needed.

The relapse generally comes on in the second or third week of assured convalescence, and in the second oftener than in the third. It is not I find—and in this the dissimilarity to the first attack is marked—preceded by the prodromic symptoms so common in typhoid fever. But abruptly, and almost without warning, the patient passes from comparative health into a decided, febrile malady; pains, furred tongue, diarrhœa, enlargement of the spleen, soon appear, and on the fourth or fifth day the characteristic rose-colored rash is seen. Now delirium manifests itself; the temperature, which has risen rapidly, remains more stationary; and the case progresses with every symptom of the first attack to a more rapid issue, convalescence being the rule. Yet even the renewed convalescence may not end the trouble. Thus I saw a case in consultation, in which the second attack terminated in rather less than two weeks, but, after an interval of about the same period, another relapse took place, and this third attack became much protracted by intense pulmonary congestion, and several violent intestinal hemorrhages happened after the sixty-sixth day of the original seizure. Fortunately,

however, the lad in whom these untoward events occurred recovered.

In these long cases there is great risk of the patient dying from sheer exhaustion; and even in less protracted cases a fatal termination may happen from the high temperature or the intestinal complication. But on the whole I am correct, I know, in stating that the chances of recovery are always far better than in the first seizure.

As regards the cause of these relapses I am unable to offer a satisfactory explanation. They are attributed to errors in diet, and in several of the cases I have seen, these seemed to have started the mishap. But errors in diet, which are not uncommon, usually occasion fresh outbreaks of diarrhœa, but not a typical fever attack, which is not a frequent occurrence. It has been ably maintained that relapses depend on a reabsorption of the morbid or typhous material thrown off from the intestinal ulcers; previously healthy glands are inoculated by the adjacent hurtful process, and the system becomes poisoned. Moreover Maclagan, who is a strong advocate of this view, contends that relapses are not met with except where there has been constipation during convalescence, and only slight diarrhœa during the first attack. As regards this point, it is contrary to my experience, and I must adopt the views expressed by Murchison in his classical treatise in opposition to it. Indeed if it be necessary to maintain that the fresh infection can only be produced if the old and unhealthy products are not swept away, clinical experience will not sustain this conclusion. In truth it will not sustain any conclusion positively or exclusively. Most likely is it that a fresh

poisoning takes place, either from redevelopment of the poison in the body, whether by the intestinal glands or elsewhere; or because part of the poison has remained latent since the first exposure, and owing to some exciting cause is called later into maturity—just as we see fresh outbreaks of malarial fever after worry or fatigue, and when there has been no chance for a new infection. To the latter view of the case I am strongly inclined. Before dismissing this part of my subject, let me say that the claims of conflicting plans of treatment have been brought forward to be adjudicated in the light of the relative frequency of the occurrence of relapses, and especially is this the case with the cold bath, the plan so vaunted by its German admirers, so eagerly credited by its opponents as being a prolific source of relapse. But as yet the data are insufficient for the settlement of this, or indeed of any question in which the comparative merits of therapeutic procedures are concerned.

Let us recur to some of the symptoms of the relapse, and inquire whether they differ from those of the primary seizure. First of the *eruption:* this, as already stated, comes on earlier; it is generally found on the fourth day, and is, as a rule, somewhat coarser and redder. It disappears on pressure, though not quite so quickly. It comes out in crops, not however with the same readiness; in other words, the early spots are more likely to last until the whole rash fades.

The appearance of the *tongue* is apt to be the same as in the first attack. Yet there are a few phenomena worthy of notice. I have often observed, indeed have learned to regard it as almost characteristic, that the tongue in typhoid fever shows at the tip a wedge

of reddish or brownish surface free from coat. This rudely triangular space becomes covered with a coat as the disorder begins to decline, or else assimilates in appearance to the general aspect of the organ. Now in the relapse this look of the tongue will also be perceived, but not generally with either equal distinctness or frequency. The same may be said of the deep transverse fissures found in severe instances of the malady.

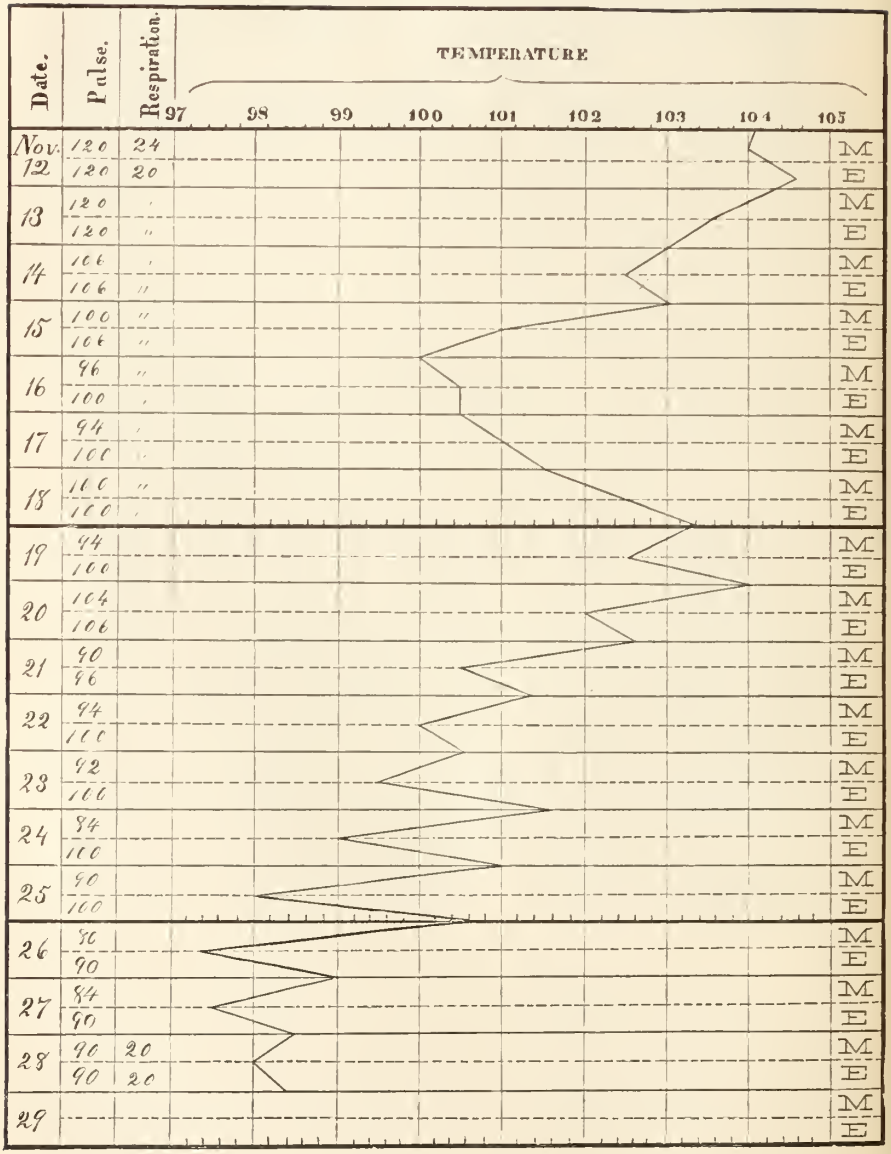
Of the other symptoms of typhoid fever, not one exists that may not be equally present in the relapse; diarrhœa, meteorism, pulmonary engorgement, delirium, present nothing unusual or different. Nor are the complications of intestinal hemorrhage or milk leg unknown. In a case that came under my notice phlegmasia dolens proved a troublesome and tedious complication. Enlargement of the spleen happens as in the original attack, and I learn from the article on typhoid fever in Ziemssen's Cyclopædia that Gerhardt makes the statement that, in many cases in which a relapse takes place, the enlarged organ is not diminished during the non-febrile period which intervenes between the original attack and the relapse.

The question of the *temperature* during the relapse has interested me much. Nor, with the exception of some allusions to it by Wunderlich, can I ascertain that it has been specially studied. It has I find these peculiarities. Unlike the graduated ascending course until the evening of the fourth or fifth day, which is the rule in ordinary instances of typhoid fever, the temperature bounds within twenty-four hours to a decided fever temperature, remits 1 to 1½° the next morning, and by the evening of the second

day is a degree or more higher than on the first day, the thermometer very commonly marking 104°. Then for from five to seven days, according to the severity of the attack, the evening figures read about the same; and a morning remission of about 1°, or somewhat more, happens, very similar to what we observe in the first attack after the initial period has passed. Subsequently occur the same more marked morning remissions and less severe evening exacerbations, until the temperature in a zigzag manner approaches to the normal, that we observe during typical cases of the typhoid attack. Yet, as here, until convalescence is established, local complications arrest or reverse the daily descent. Neither do we always find during the height of the relapse, that the temperature is as regular as described. It may sink almost continuously for the first three days after it has reached the height occasioned by the returning fever, and then for three or four days more gradually ascend without any morning remission, yet subsequently, as defervescence sets in, show the characteristic zigzag decline alluded to. All this was markedly shown in a case under my care at the Pennsylvania Hospital; as, moreover, this case will tend to illustrate some other matters to which I wish to call attention, I shall report it in some detail.

A German porter, twenty-five years of age, was admitted into the hospital on October 17, with well-marked typhoid fever, and occupied Bed 16, Men's Medical Ward. He had been ill for several weeks, and presented a temperature of 103½°, a flushed face, marked tremor, severe diarrhœa, tympanitic abdomen, and a copious eruption of lenticular spots. He made a slow convalescence, the tremor remaining after

the fever had left; but by November 2, he had scarcely any trembling; his voice, which he had lost from weakness, returned; he had no longer looseness of the bowels, and the temperature was normal. On the 7th, the temperature having for several days marked 98° in the evening, the record was discontinued. On the 11th, the patient walked about the wards, talked a good deal with visitors, wrote a long letter, was evidently much fatigued, and by evening had a temperature of 104°, and a pulse of 120. The next morning it was most evident that a relapse had taken place; the tongue became coated at the sides, and subsequently dry, brown, and fissured; the characteristic red spots appeared on the chest and abdomen, on or rather just before the fourth day of the relapse; delirium and restlessness became marked features; indeed most of the symptoms of the first attack were reproduced. Yet not as regards the abdominal symptoms; for the bowels remained regular, or were only occasionally loose. By the 26th, the dryness of tongue had disappeared, only traces of spots could be seen; the face was still flushed, but pulse, respiration, and temperature denoted convalescence. What the changes in this symptom were need not be specified; the accompanying table (page 108) shows them at a glance. But there is a further symptom which exhibited itself plainly during the weeks in December in which the patient remained in the hospital gaining flesh and strength, namely, distinct lines across the nails; the first line the outgrowth of the original attack, the second slowly following the relapse, and both together giving the distinctive markings reproduced in the plate.


Date.
Pulse.
Respiration.
TEMPERATURE
97
98
99
100
101
102
103
104
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Nov. 12
120 / 120
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106 / 106
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Let me in conclusion say a few words more about the appearance of the nails, and the lessons it may teach. My attention was called to the changes the nails undergo in fevers long ago by Dr. Longstreth, and as he will present a paper to-night examining these changes in detail, I shall not attempt to describe them minutely. But I will point out here that with the relapse of typhoid fever the second ridge of the altered nail-growth comes to tell us how completely in every respect the fever has been reproduced; and the first ridge may in obscure cases give us the true meaning of doubtful symptoms, and prove conclusive of the diagnosis. A case in point happened during last winter at the Pennsylvania Hospital.

A boy, thirteen years of age, was sent to Bed 12 of my ward, December 6th, of last year, and during my temporary absence Dr. Hutchinson for a few days kindly took charge of him. The history was most unsatisfactory. He had received a blow on the back of the head about a month before admission, and had had a cough for several months prior to this accident. He had been confined latterly to bed for three weeks; had bled from the nose once; and during the first week had had a diarrhœa, which was readily checked by medicine. The boy, though he was stated to have been delirious for a week, and showed some hebetude, answered questions intelligently. He complained of great weakness, pain in the bowels, and tenderness in the muscles of the lower extremities. He had sordes on the teeth; shallow, frequent respiration; râles in the chest, some fine; a pulse of 120, readily compressible; and an evening temperature of 105°. He soon became extremely delirious; he was very weak; his feet were cold; indeed his condition was so grave that recovery was regarded as very doubtful, and, notwithstanding the obscurity of the symptoms, wine, chloric ether, and other stimulants were freely resorted to. Some of the symptoms,

and the history, pointed to a brain trouble, but the case was regarded as one of typhoid fever; and its progress, the tympany, the occasional diarrhœa, the tenderness at the lower part of the abdomen, the look of the tongue, the course the chest symptoms took, and the markedly typhoid aspect of the face, rendered this opinion more and more certain, although no eruption except sudamina could be found. But what gave us the most information, and told us that we were really dealing with a second attack, thus explaining the extraordinary length of the malady—a length with difficulty reconcilable to the view of ordinary typhoid fever—was that on the 17th it was noted that the nails about half way up showed a white line of impaired nutrition, evidently the result of the first attack of illness, and that near the root another white line was developing, due to the relapse.[1] The

[1] I showed the case to Dr. Longstreth, and he was kind enough to hand me a minute record of the condition of the nails taken (Jan. 29, 1877) shortly before the patient left the hospital.

Hand.—The nails of all the fingers show the peculiar changes; the middle and ring finger nails of the left hand most distinctly. This is a description of the left middle finger nail: Behind the free extremity of the nail there is a narrow, pinkish area extending across the nail from side to side, convex anteriorly. This part is normal, and has not been affected by the nutritive changes incident to the fever. It formed, at the commencement of the illness, the part of the nail situated near the posterior part. Behind this area is a whitish streak; its anterior border fades off into the normally colored nail in front; its posterior border is pretty sharply defined. Posterior to this whitish streak comes a very narrow area of pinkish-colored, normal nail, fibre-like in width, and which can be seen only by very close inspection. This second area of normal nail is about midway from the cut border to the root. These changes represent the alterations which occurred previous to the patient's admission into the hospital. The nail at this part shows no furrow or ridge. The portion of the nail behind this second normal area exhibits the alterations the progress of which I have watched during his residence in the ward. The effects are much more distinctly marked than the changes just described; there is to be seen not only the anæmic streak but also a change of level extending from side to side of the nails.

The second series of changes commences with a broader whitish or anæmic streak than the anterior whitish streak; the color is of a deeper white, and this part, viewed with oblique illumination, lacks the lustre and polish of a normal nail. A profile view shows a depression of the level of the nail,

patient made a very slow recovery, and was not free from fever until the 26th. When quite himself, we learned from

which, commencing within the limits of the white streak, slopes downwards and backwards towards the root of the nail. At the back of the slope the tissue of the nail rises abruptly to the normal level. Its direction is nearly straight across. The posterior border of the depression is just protruded from beneath the fold of skin covering the root, and lies within the area of the lunula. The depth of the depression is greater in the middle of the nail than at the borders. In the depressed area the longitudinal striæ of the nail are more prominent than normal. In this second series there are two parts to be distinguished, viz., the depression, and the piece of nail anterior to it, which is anæmic and without lustre, but the level of which corresponds to that of normal nail. During the fever-process the area of depression corresponded to the root of the nail; the whitish nail in front of the depression was then covered by the anterior margin of the fold of skin at the root.

On each thumb-nail the first series of changes are to be seen distinctly, and they resemble the appearances already described. Of the second series of changes, only the anæmic streak can be seen; the depression is still covered by the fold of skin at the root. The slower growth of the thumb-nail accounts for the difference between it and the other nails. (A month later the depression was distinctly to be seen on the thumb-nails, convex anteriorly. On the other fingers the first series of changes had grown forward and had been cut off, whilst the second series of changes had advanced close to the free extremities of the nails.)

Feet.—Similarly on the toe-nails are seen the nutritive changes. The matrix is very short; the greater part of the nail is unattached. At the commencement of the examination, the skin at the root is stretched forward and is adherent to the dorsum of the nails. In front of the adherent skin is a black line; the surface of the nail is rough along this line, and dirt has colored it black. After washing the foot in hot water, the adherent skin is easily removed, and the black line nearly disappears. There is now seen at the place where the black line was, a very narrow whitish or anæmic streak, fibre-like in width, and without lustre. This anæmic line represents the first series of changes, and corresponds to the anterior markings on the finger-nails. The second series of alterations commence behind the anterior anæmic streak, and are separated from it by a very narrow area. After the removal of the epidermis is seen a whitish area (it is within the lunula, but it is whiter than the normal lunula) which is depressed markedly beneath the level of the normal part of the nail. The depression continues under the skin, but by tearing off the skin and exposing part of the root, the ridge terminating the depression or furrow is found. This area is of a dead white color, without lustre, and uneven. The other toe-nails, the growth of which is more rapid than that of the great toe, show the ridge and depression without tearing

him that he had had unmistakable signs of typhoid fever for several weeks before his admission; that he had been treated for typhoid fever by a physician; and that he was rapidly getting well when the untoward symptoms arose which sent him to the hospital.

It is needless to say of how much value in this case the study of the nail-growth proved; how it alone gave the full clue to the case; and how we may hope by this and similar investigations to add much needed information to our knowledge of the relapses of continued fevers.

away the skin covering the root. The area occupied by the alterations in the toe-nails is much less than in those of the fingers, owing to the slower growth of the former. By passing one's finger-nail over the dorsum of the patient's nail, the alterations of level can in some cases be more distinctly felt than they can be seen.

ON

CHANGES IN THE NAILS IN FEVER, AND ESPECIALLY IN RELAPSING FEVER.

By

MORRIS LONGSTRETH, M.D.,

PHYSICIAN FOR OUT-PATIENTS, AND PATHOLOGIST AND CURATOR, TO THE PENNSYLVANIA HOSPITAL.

[Communicated by Professor Da Costa.]

[Read June 6, 1877.]

The first time that my attention was attracted to alterations in the growth of the nails, other than those produced by direct violence to the matrix, or by inflammation of the soft parts of the finger or toe,[1] was in the spring of the year 1869. The observation was made on my own person, after an attack of Relapsing Fever. The accession of the fever was sudden; the primary paroxysm lasted seven days, the intermission ended on the fourteenth day, the relapse continued seven days longer, and ended with a very profuse sweating on the twenty-first day of the disease. The fever-periods began with violent headache, soon followed by delirium which was continuous during both the primary paroxysm and the relapse.

At the time my observations were made, there were but two records in medical literature bearing on this

[1] I may mention, also, that I have seen somewhat similar changes in the nails after fractures of the long bones of the extremities.

subject, viz.: Reil's (*Memorabilia Clinicorum*, Fasciculi. 3, p. 206; Halæ, 1792), and Beau's (*Archives Générales de Médecine*, 1e ser., tome xi., Août, 1846, p. 447), both of which were unknown to me. In June, 1870, Vogel published an article in the *Deutsches Archiv für klinische Medicin*, on the Nails after Febrile Diseases (Die Nägel nach fieberhaften Krankheiten), in which he gives the history of the subject, the characteristics of the nail changes, and the diseases in which he has seen the changes, especially Typhus. I was not acquainted with these observations until Professor Da Costa showed me Vogel's paper within a few weeks of the present time, and also the allusion to the subject in Murchison on Fevers; since then I have seen the original papers of Reil and Beau. I have not been able to find any further references.

My examination extended to the nails of my hands only, and I have no knowledge of the condition of my toe-nails. The appearances were nearly uniform on all, so that a description of one nail will suffice; the characteristic changes had approached nearer the free margin in some nails than in others, owing to the different rates of growth. The differences of distance were not marked, although distinct; I have no record of the measurements of the nails.

There were to be seen distinctly on the face of each nail two concentric lines (arcs of a circle), extending from side to side of the nail substance, having their convexity forwards, and nearly the same curve as the free margin of the nail. The color of the lines was white; nearly as dense a whiteness as is seen in the milky dots and streaks which occur so frequently in

some finger-nails from bruises, etc. These lines were narrow, equalling in width, each of them, coarsely ruled lines, *e. g.* ▬. The interval between them was about the same width, possibly a little broader than each line taken separately. The normal, longitudinal striæ were not distorted, and were certainly not more distinct than normal, as I have since then seen them in the nails of patients with these peculiar markings due to febrile, or otherwise-changed, nutritive processes. The borders or margins of the white lines were not absolutely, sharply defined, as would be the ruled line to which I have compared them, but very slightly shaded off into the adjacent, normal-colored nail-substances. The shading off was very slight, however; much less in amount than I have since seen it in cases of typhoid fever. It was rather to be compared to the dispersion seen at the borders of an ink line made with a pen on slightly absorbent paper, as compared with such a line made on ordinary, glazed, writing paper. The space between the two lines had the color of the normal nail in advance of and behind the two lines, that is to say, of parts of the nail developed during health, previous to and subsequent to the attack of fever.

When I first observed the peculiar markings on my nails, that is to say, when the seat of the markings had been advanced by protrusion beyond the lunula—for whilst the white lines were yet within the lunula their white color so nearly corresponded to the normal coloration of this part as to attract no attention—there was a loss of the surface-lustre normally seen on nails. This loss of lustre was confined to the neighborhood or direct seat of the white lines themselves.

As the lines were protruded towards the free margins of the nail, by growth, the polish was restored. It is probable that friction on the nails from continual rubbing reproduced the usual lustre, as seen in oblique illumination. There was never visible any change in the form of my nails; there was no depression or furrow at or near the seat of the lines; there certainly were no such furrows as I since have noticed following the nutritive interruptions of typhoid and other febrile processes, where it is possible not only to see, but also to feel, the depression in the level of the nail.

When the white lines had advanced to the free margin of the nail, and were cut off, the nail-substance was found much more brittle than normal. In paring the nail with a knife, the tissue splintered in flakes, and left the border irregular and rough, unlike the smoothly cut end of a normal nail. There was also an unusual tendency for the nails to break from forcible contact with objects.

After my recovery and return to duty at the Pennsylvania Hospital, I had under my care many cases of Relapsing Fever. After noticing these peculiar markings on my own nails, it was natural to turn to the finger-nails of my patients, to see if they bore similar tracings of their illness. Here, too, I found the markings resembling those on my own fingers. The patients, with one exception, had each suffered one relapse, and the two lines were to be distinctly seen, possessing similar characteristics to those already described. The one exception, above mentioned, had had two relapses, and in this case I found three distinct white lines on the finger-nails. The two lines

nearest the free margin of the nail were as distinctly marked in this as in any of the other cases, whilst the third, the last produced line, was of a fainter color, and seemed more diffuse and less sharply defined; this line was, however, near the lunula, to the color of which these white streaks so closely correspond, and to this fact may be due the less marked distinctness of this than that of the two more advanced lines. The patient was soon discharged from the hospital, and passed out of my observation.

Professor Alfred Vogel gives in his article the details of certain cases of typhus, with the changes observed in the nails, together with several colored plates of the characteristic appearances. All of the figures refer to periods after the fever has ceased and the nails have resumed their growth, the markings having been protruded beyond the lunula, and approaching the free margin of the nail. Manifestly, therefore, as he says himself, nail changes have had to him a value for determining the pre-existence of the fever process, or for its exclusion. The nail changes, as he describes them in typhus fever, and as I have described them in relapsing fever, have no significance until the febrile stage is passed, and the protrusion of the nail comes by growth. Vogel makes no mention of relapses, and consequently no mention of changes coming as a result of the second febrile process. "But since in typhus one and the same individual never has twice quickly one attack after another, so is the possibility of such an exclusion of great diagnostic worth upon the admission of a fever patient, in whom these traces of a passed typhus are discovered" (Vogel). This sentence shows clearly that Vogel

has not made observations on nails where are to be seen such changes as would clearly indicate the past occurrence of a relapse of the fever process.

Reil, likewise, in the fifty or sixty words which he devotes to the subject, says nothing of the effect of relapses in producing nail changes. Beau does not speak of relapses in any febrile process—all of such processes being, as he says, capable of producing the changes; he makes allusion to a case of acute rheumatism, seen in March, 1846, the patient having been ill in the previous June with severe typhoid fever; the nails of the great toes, and those only, presented the markings of the typhoid fever, whilst all the nails bore the more recent traces of the acute rheumatism. Murchison (2d edition, 1873) makes a very short allusion to the subject without any description of the changes in the nails, although he quotes Vogel's article, and gives a wood-cut of his plate.

Whilst, as is evident, these peculiar markings are of value in their indications of past events, is there nothing to be observed in the nails, which, at the commencement of and during the continuance of the fever, is of significance; or do we have to wait until the growth of the nail is resumed, and the white marking, as the trace of the fever process, has been protruded beyond the root and lunula? During the last four months, through the kindness of Drs. Da Costa and Hutchinson, I have been able to carefully observe during the febrile movement the appearances presented by the nails of many patients, especially the parts near the root, and also the relation which the epidermis has borne to the root of the nail. Here I have found a constant peculiarity.

Let me note the appearances that were seen. In the man who occupied Bed 16, during the relapse that occurred, in the very first days after the temperature had risen, the skin at the root, instead of separating from the surface of the nail as it does in normal growth, maintained its adherence; in the latter days of the fever-relapse, the skin was seen to be stretched forwards on the surface of the nail to the extent of more than a line. In other words, the epidermal layer of the skin around the root became adherent to the horny nail, and was stretched forwards, thus deepening the fold, at least as far as the epidermal layer was concerned, and covering the horny nail farther in advance of the root (by about a line) than is seen under normal conditions. The appearance is similar to that which any one may see occasionally on his own nail, or may produce by scraping with a knife deeply under the free edge of the posterior fold. This scraping in some way leads to adhesion of the epidermic layer of the skin on the posterior part of the nail; this layer, adhering and, as the nail grows, being carried forward and becoming stretched on the dorsum of the nail, covers the area of the lunula. By further growth (after the scraping experiment) this adhering, epidermal layer is first separated from its skin-attachments, and lastly is separated from the horny nail, leaving the surface which was scraped, rough or lustreless. In the patient of Bed 16, the markings on the nail from the primary paroxysm of fever were seen in advance of the adhering skin during all the period of the relapse; they were within the area of the lunula, and therefore less distinct than subsequently.

The same condition of adherence was present in

the boy who occupied Bed 12, and this condition, together with the existence directly in advance of it of the white, anæmic streak of nail, confirmed the suspicion that the patient was suffering from a distinct relapse of the fever, and that we were not dealing with the primary paroxysm.

In some cases of typhoid fever which I have seen lately, during the existence of the febrile movement, the condition of the skin around the root of the nail differed from that which I have described as usual. The patients to whom I refer were sailors, and in them I found that, from the greater hardness and dryness of skin incident to their manual labor, the epidermis was not adherent, but cracking and separating around the root of the nail. The condition of the nail underneath the projecting, but cracked and not adherent, skin, was similar to what I have found in other cases in which I have artificially removed the adherent epidermic layer, *i. e.*, the surface was rough and appeared to have a membrane adhering to it. In other words, the nail was not perfectly developed, and, although it had become hard, had not become smooth, polished, and truly horny. Thus by carefully noting the adhesion of the skin to the nail, or the condition of the nail when such adhesion has not occurred, we have the means, independently of the phenomena of fever, of determining the occurrence of checked nutrition.

Therefore, adhesion of the epidermis to the nails on all the digits, as I have described it, or the appearance as I saw it in the sailors referred to, taken in conjunction with a white line or furrow, would, I

take it, be diagnostic of a relapse of fever or other process which interfered markedly with nutrition.

Finally, it remains to speak of the minute changes in the nutrition of the nails which lead to the varied appearances presented by them. We have to account for the occurrence of loss of lustre, and for that of the white or anæmic[1] streak and of the furrow or ridge[2] that commonly make their appearance. The latter two changes I consider to be identical; the one or the other, or perhaps both, result from the varying severity of the nutritive disturbance, and are, therefore, to be looked upon merely as differences of degree in one and the same process.

Let us consider first the loss of lustre. This diminution of the physiological, mother-of-pearl appearance of the nail is, according to my observation, seen in advance of the anæmic streak, *i. e.*, nearest the free extremity of the nail. It never continues to exist very long, for continual use soon restores the normal polish of the surface. The loss of polish extends also backward towards the root, until it reaches the part

[1] I do not consider that *anæmic* is the proper word to apply to this condition; the whiteness of the streak is not due to local anæmia, but to an opaqueness resulting from tissue-changes. The only connection which anæmia can have with the appearance, is that a general (systemic) anæmia exists at the time at which the horny scales of the nail (afterwards showing white) are newly formed at the posterior border of the nail.

[2] The "ridge," in all the cases which I have seen, was *not* produced by changes taken place during the fever-process, and it is, therefore, not to be included in the area of the fever-changes on the nail. This is not a ridge, *i. e.*, there is no elevation above the general normal level. The edges of the normal nail, bordering the furrow in front and behind, are "ridges" in comparison with the depth of the furrow. A ridge, such as Vogel pictures, rising above the level of the normal parts of the nail, I should regard as the result of a local nail-disease, *e. g.*, inflammation, and not as a local change due to a systemic disturbance of nutrition.

of the nail developed normally, subsequently to the febrile process. That is, this change extends over all that portion of the nail which exists as the root, or is concealed beneath the fold of skin at its posterior part, during the period of nutritive disturbance. The lustreless part, in advance of the anæmic streak, corresponds to the area to which the epidermis adheres during the febrile process, or to the part which, when the epidermis does not thus adhere, appears rough, as though covered with a membrane.

Vogel, in the paper already quoted, does not allude to the occurrence of any loss of lustre extending in advance of the anæmic streak; perhaps because of its temporary nature. Since commencing to write this description, I have had a very favorable opportunity for watching the development and progress of these nail changes. In the case here specially alluded to, one of brain disease with only slight febrile movement, but with profound disturbance of nutrition, the nails now present not only the white streak and loss of lustre, but also a deep furrow. During convalescence, which was very protracted, the patient was so feeble that the hands were little used, and I had the opportunity of confirming my previous positive observations, that a marked loss of lustre existed in advance of the white line. This loss of lustre remained visible until it advanced to the free extremity and was cut off. It remained visible thus for an unusually long time, because the nails were not polished by the continual attrition of use. I may here also mention that, during the convalescence of this patient, a slight relapse occurred, and immediately adhesion of the epidermis near the root of the nail took place, and the

skin here became slightly stretched forwards. The part of the nail to which the skin was observed to adhere at that time, is still within the lunula, so that I am unable to speak of the occurrence of loss of lustre, or of the production of a second anæmic streak; and, indeed, considering the very short duration of the relapse, it is questionable if any such effects will hereafter be observed.

The furrow and the whitish or anæmic streak are, as I have said, identical; they are the results of the same process, the different appearances being merely differences of degree in the nutritive alterations or disturbances. Vogel in his article, quoting from Reil, speaks of the complete falling off of the nails seen after malignant fevers. This result is of course to be regarded as the effect of a more violent disturbance of the nutritive processes, which shows itself by destroying cell-growth not only at the root of the nail, but also throughout the whole of the matrix.

The anæmic streak is the least marked result of such changes; possibly also, though of this I am not able to speak from my own observation, a simple loss of polish may be the least marked result in the milder cases. Such an appearance, however, from its exceedingly transitory nature, can be of little value in a diagnostic point of view.

The streak and the furrow, it is self-evident, are results of changes in growth taking place in the root of the nail during the febrile movement. The cells of the rete Malpighii, in the fold and at the root of the nail, are checked in the growth, and illy formed. The growth of the cells is not completely stopped, but the tissues never reach perfection. The white

line differs from the furrow in that in the former the cells of the horny layer are merely opaque, whilst in the latter there is observed not only opaqueness, but also an actual decrease in the normal size and number of cells. The condition of the part at the fold during the fever, and the appearances presented by the nail subsequently, indicate that the growth is not wholly checked by any febrile disease. The adhesion of the skin and the stretching of the epidermal layer contradict such a supposition. If growth ceased completely, the nails after every fever-process would present an absence of substance, a loss of continuity at the part representing the root of the nail during the existence of the nutritive disturbance. We must therefore conclude that some degree both of growth and of protrusion—that gradual sliding of the nail forward over the matrix—continues.

The growth of the nail during the early part of the fever, and in the latter part, varies often very considerably, as the indications distinctly show. For example, what are the appearances presented by the nails in an uncomplicated case of typhoid fever in a moderately severe form? In the cases which I have observed, there have been seen, nearest the free extremity of the nail, first the area of loss of lustre, then behind this the whitish streak, and behind this again the furrow. The nail, however, from behind the lustreless area has been depressed, the depression attaining the maximum in the furrow. From the depth of the furrow the nail has remounted more or less abruptly to the normal level, and has resumed the usual tint. The longitudinal striæ have been distorted in the area of the depression, and the nail

in this area has been withal narrower than in the normal condition. These changes have corresponded with the fever history. The accession of the attack has been gradual. There has been adhesion of the epidermis, leaving as its trace a loss of lustre in advance of the white line. The new elements at the posterior border of the root have been slightly less developed, resulting in an opaqueness or whiteness. In the early part of the fever, growth has not ceased —note the stretching of the epidermal layer—the horny lamina has been pushed forward over the softer layer of cells beneath it, by the constant production, although imperfect formation, of the new elements which, at the back of the root, have been becoming horny scales. Towards the end of the fever-period, when the nutritive disturbances have become more profound, the development of the new-forming elements at the posterior border of the root has been more interfered with, resulting in a loss of substance. When the fever has ended, the normal growth of the nail has been resumed.

In other cases which I have seen (for example a case of acute rheumatism), the onset has been sudden and severe; the nails have presented the loss of lustre in advance; directly behind this has come the furrow, commencing with an initial, abrupt descent, having a variable width, and then gradually remounting to the normal level in correspondence with the gradual defervescence.

REPORT

OF THE

COMMITTEE ON METEOROLOGY AND EPIDEMICS FOR THE YEAR 1876.

By

RICHARD A. CLEEMANN, M.D.

[Read June 6, 1877.]

The meteorological part of the Report which I have the honor of presenting to the College this evening, is compiled from the records of the United States Signal Service for this city, to which I am courteously permitted access. That portion which refers to epidemics has been prepared from the mortuary lists of the City Registrar, Mr. Geo. E. Chambers, who has kindly allowed me to use those for last year in advance of their publication. I have also consulted the Reports of the Board of Health of Philadelphia for several years past, of late so carefully and intelligently edited by the Secretary, Wm. H. Ford, M.D.

Meteorology.

Temperature.—The mean temperature (Table A) of the year 1876 was 52.6° Fahr., nearly one degree above the average for the last five years (1872–1876), 51.7° (Table B). The excess was widely distributed, for all the months, excepting two (September and December), contributed to this increase. But January was much

TABLE A.—METEOROLOGICAL ELEMENTS OF THE CLIMATE AT PHILADELPHIA FOR 1876.

Furnished by F. M. M. BEALL, Observer, Signal Service, U. S. A.

Date.	Mean humidity.	Barometer.							Thermometer.							Wind.							Am't of rain and melted snow.		No. of auroras.
		Mean	Means of			Range.			Means of				Range.			Prevailing direction.	Number of miles.						Amount in inches.	No. of days on which rain or snow fell.	
			A. M. observations.	P. M. observations.	Night observations.	Highest.	Lowest.	Difference.	Mean.	A. M. observations.	P. M. observations.	Night observations.	Maximum.	Minimum.	Difference.		Noon to 6 P. M.	6 P. M. to midnight.	Midnight to 6 A. M.	6 A. M. to noon.	Max. veloc. during hour.	Total.			
Jan.	70	30.161	30.186	30.135	30.161	30.614	29.534	1.08	37.7	34.8	41.2	36.3	67	15	52	S. W.	2462	1674	1797	2125	44	8358	1.52	10	None observed.
Feb.	69	30.131	30.152	30.121	30.129	30.964	29.029	1.93	34.0	31.1	38.1	33.4	62	6	56	N. W.	2363	1717	1786	2253	48	8119	5.03	16	"
March,	68	30.040	30.100	30.012	30.032	30.540	29.165	1.37	37.4	33.7	41.5	36.1	66	9	57	N. W.	3051	2494	2109	2613	46	10264	6.74	11	"
April,	53	30.001	30.043	29.9.3	30.001	30.524	29.543	0.98	49.1	45 5	55.5	46.2	74	30	44	N.	2777	2046	1776	2247	40	8846	2.16	11	"
May,	62	30.054	30.090	30.012	30.0 34	30.451	29.663	0.79	60.9	56.6	69.7	58.2	85	38	47	S. W.	2401	1679	1443	2016	34	7539	4.45	9	"
June,	65	29.998	30.028	29.956	29.9.9	30.218	29.701	0.51	74.2	70.7	80.6	70.6	93	52	41	S. W.	2010	1386	1016	1435	30	5847	2.29	10	"
July,	63	29.993	30.026	29.953	29.99.3	30.274	29.767	0.51	78.6	75.5	84.4	75.4	100	60	40	W.	2087	1508	1186	1745	40	6526	5.74	11	"
Aug.	69	30.061	30.087	30.027	30.065	30.241	29.803	0.44	74.3	70.5	80.4	71.8	91	56	35	E.	4674	1236	1110	1405	24	5452	0.98	8	"
Sept.	71	29.984	30.001	29.948	29.992	30.281	29.566	0.71	63.8	61.0	68.4	61.9	88	46	42	E.	2399	1777	1754	2213	52	8143	8.77	15	"
Oct.	65	30.029	30.049	29.98.9	30.030	30.454	29.573	0.88	50.8	46.4	56.3	48.4	75	31	44	N. W.	2210	1482	1464	2013	40	7169	1.06	8	"
Nov.	71	29 969	30.005	29.943	29.969	30.395	29.541	0.85	45.0	41.8	47.8	43.6	77	23	54	E.	2265	1821	1834	2191	48	8111	7.31	12	"
Dec.	73	30.027	30.036	30.012	30.038	30.693	29.037	1.66	25.5	22.9	27.9	24.4	49	4	45	N. W.	2546	2228	1994	2468	42	9206	1.40	10	"
Ann. mean	66.6	30.037	30.067	30.005	30.040	30.470	29.494	0.97	52.6	49.2	57 6	50.5	77.2	30.1	46.4	W.	....	....	....	....	40.7	....	...	....	
Total,	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	....	28,212	21,375	19,269	24,724	..	93,580	47.39	131	

above the average, the record showing 37.7° against the mean 31.8°; as February also ranged high, 34° against 30.9°, the winter was exceptionally mild. The spring was somewhat warmer than usual, for while March and May were each a fraction above their average temperature, April exceeded hers by 2.3°. The thermometer in June was quite high, 74.2° against the average of five years, 72.4°; again, in July it read 78.6° against 76.6°, and in August 74.3° against 73.2°: these figures indicate a summer as warm as the remarkable one of 1872. But in September the temperature fell 2.1° below the mean, rising again in October 0.5° above, and 3.9° above it in November; these variations, with other meteorological conditions to be considered further on, giving the fall an unusual constitution. December was very cold, marking only 25.5°, which is a little more than 7° below the mean: there were twenty-one days in this month in which the thermometer did not rise above the thawing point. It is the low temperature of December which makes the unusual heat of other portions of the year less perceptible in the annual average.

The range of the thermometer for the whole year was through ninety-six degrees, from the points 100° in the month of July to 4° noted in December. The months placed according to the extent of their special ranges of temperature read March 57°, February 56°, November 54°, January 52°, May 47°, December 45°, April and October, each 44°, September 42°, June 41°, July 40°, August 35°. This, excepting the position of November, which should be the seventh instead of the third on the list, is very near the usual sequence, the weather being more unsettled in the early spring and in the

colder months. The range in November was 8° more than its average for five years, that in July 6° and that in March 5° more, while December, on the other hand, ranged 4° less. The greater variation in July took place in the last week, the daily mean of the thermometer for each day of the first three weeks having never fallen below 79°: this extraordinary succession of hot days was part of an unparalleled series which began on the 24th day of June. Frost was observed as early as the 26th of September, and the first ice formed the 16th of October.

Pressure.—The mean reading of the barometer for 1876 is 30.037 inches, 0.017 of an inch below the average for the last five years. The lowest point reached during the year by the level of the mercury was 29.029 inches, in the month of February; and the highest 30.964 inches, in the same month; a range of 1.93 inches. The next lower and higher points were noted in December, 29.037 inches and 30.693 inches respectively, a variation of 1.66 inches. The order of the months in regard to height of mean pressure was January, 30.161 inches; February, 30.131 inches; August, 30.161 inches; May, 30.054 inches; March, 30.040 inches; December, 30.027 inches; October, 30.020 inches; April, 30.004 inches; June, 29.998 inches; July, 29.993 inches; September, 29.984 inches; November, 29.969 inches. Thus, generally speaking, the barometric pressure may be said to have been greatest in the winter, next in the spring, then in the summer, and least in the fall: nevertheless there were striking departures from the rule in individual months. In extent of range, the winter came first, then the

spring, the summer, and finally the fall: in the last season the variation was only 0.57 inch.

Rainfall.—The amount of rain and melted snow recorded as falling in 1876 was 47.39 inches, about equal to the average for the last five years: it was gathered on 131 days, eleven less than the annual mean of wet and snowy days. The months in which the precipitation was greatest were September, 8.77 inches (twice its average); November, 7.31 inches; March, 6.71 inches (again twice the average), and July, 5.71 inches: much less amounts were noted in August, 0.98 inch (only $\frac{1}{7}$ the average); October, 1.06 inches ($\frac{1}{3}$ the average); December, 1.40 inches; January, 1.52 inches, and April, 2.16 inches (the last three months diminished by half their averages). The small rainfall of August is very remarkable, since this part of the year is in general distinguished by very heavy showers, more rain falling than in any other month: the drought was compensated for, as we have seen, by the doubling of the average amount of rainfall in September: on a single day, the 17th of this month, $3\frac{1}{2}$ inches of rain fell.

Humidity.—The mean humidity of the atmosphere for the year 1876 is expressed as 66.6°, saturation being 100°: and differs little from the average of five years. The most humid months were December, 73°; November and September, each 71°; January, 70°; February and August, each 69°: the least, April, only 53°; May, 62°; July, 63°; and October, 65°. The most noteworthy facts implied in the above figures are the dryness of April, in humidity 5° below the average for four years, and the opposite as regards May and November, the former month marking 4°

TABLE B.—COMPARISON OF METEOROLOGICAL ELEMENTS OF THE CLIMATE AT PHILADELPHIA, FOR THE YEARS 1872, 1873, 1874, 1875, AND 1876, WITH AVERAGES.

Meteorological elements.	Month	1872.	1873.	1874.	1875.	1876.	Mean of 5 years.
Thermometer. Mean.	January.	29.4	28.9	36.7	26.1	37.7	31.8
Thermometer. Range.	January.	43.0	60.0	55.5	49.5	52.0	52.0
Mean barometer.	January.	30.053	30.113	30 193	30.231	30.161	30.150
Mean humidity.	January.	.....	.....	70.7	71.0	70.0	70.4
Rainfall.	January.	.95	5.84	4.53	2.83	1.52	3.14
Thermometer. Mean.	February.	31.2	29.8	33 2	26.1	34.0	30.9
Thermometer. Range.	February.	45.0	50.0	64.0	58.0	56 0	54 0
Mean barometer.	February.	30.011	30.016	30.174	30.131	30.131	30.092
Mean humidity.	February.	.....	.....	67.7	66.0	69.0	67.0
Rainfall.	February.	1.12	4.75	2.46	3.20	5.03	3.31
Thermometer. Mean.	March.	32.1	39.1	40.0	34.9	37.4	36.7
Thermometer. Range.	March.	52.0	53.0	48.0	49.0	57.0	52.0
Mean barometer.	March.	30.054	30.001	30.022	30.104	30.040	30.056
Mean humidity.	March.	.....	.....	64.0	69.0	68.0	67 0
Rainfall.	March.	3.67	2.04	2.16	3.10	6.71	3.53
Thermometer. Mean.	April.	51.6	45.3	42.5	45.5	49.1	46.8
Thermometer. Range.	April.	54.0	35.0	46.5	51.0	44.0	46.0
Mean barometer.	April.	30.063	29.947	30 055	29.978	30.004	30.009
Mean humidity.	April.	.....	47.5	72.2	59.0	53.0	58.0
Rainfall.	April.	2.60	3.51	9.76	2.85	2.16	4.17
Thermometer. Mean	May.	61.5	59.6	59.9	61.4	60.9	60.7
Thermometer. Range.	May.	43.0	45.0	51.0	49.0	47.0	47.0
Mean barometer.	May.	29.975	30.009	29.982	29.993	30.064	30.004
Mean humidity.	May.	.....	60.3	57.7	54.0	62.0	58.0
Rainfall.	May.	2.15	5.83	2.75	1 36	4.45	3.31
Thermometer. Mean.	June.	72.7	70.6	73.0	70.5	74.2	72.4
Thermometer. Range.	June.	36.0	43.0	46.0	44.0	41.0	42.0
Mean barometer.	June.	29.976	29.996	29.990	30.034	29.999	29.999
Mean humidity.	June.	.....	63.0	64.1	65.0	65.0	64.0
Rainfall.	June.	[4.29	0.90	3.02	4.13	2.29	2.91
Thermometer. Mean.	July.	78.6	76.4	74.7	74.6	78.6	76.6
Thermometer. Range.	July.	28.0	33.0	31.0	31.0	40.0	34.0
Mean barometer.	July.	29.970	30.023	30.030	29.996	29.993	30.010
Mean humidity.	July.	.....	62.7	64.1	66.0	63.0	64.0
Rainfall.	July.	9.20	5.00	2.25	3.63	5.71	5.16

TABLE B—*continued.*

Meteorological elements.	M'nth	1872.	1873.	1874.	1875.	1876.	Mean of 5 years.
Thermometer. Mean.	August.	75.9	72.3	71.1	72.4	74.3	73.2
Thermometer. Range.		34.0	33.0	40.0	27.0	35.0	34.0
Mean barometer.		30.045	30.065	30 030	30.035	30.061	30.047
Mean humidity.		.....	75.4	61.3	76 0	69.0	70.0
Rainfall.		7.81	11.49	5.65	6.42	0.98	6.47
Thermometer. Mean.	September.	67.5	66.1	68.0	64.1	63.8	65.9
Thermometer. Range.		40.0	43.0	36 0	45.0	42.0	41.0
Mean barometer.		30.049	30.096	30.100	30.035	29.984	30.053
Mean humidity.		.....	70.6	71.0	65.0	71.0	69.0
Rainfall.		3.66	3.58	6.01	2.53	8.77	4.51
Thermometer. Mean.	October.	52.9	54.7	55.5	53.7	54.8	54.3
Thermometer. Range.		38.0	43.5	42.0	39.0	44.0	41.0
Mean barometer.		30.105	30.073	30.115	30.015	30.020	30.065
Mean humidity.		.....	63.4	65.0	65.0	65.0	64.0
Rainfall.		5.20	5.20	2.87	1.42	1.06	3.15
Thermometer. Mean.	November.	41.2	38.3	41.8	39.7	45.0	41.1
Thermometer. Range.		42.0	35.0	45.0	54.0	54.0	46.0
Mean barometer.		30.099	29.992	30.200	30.107	29.969	30.073
Mean humidity.		.....	63.9	61.0	68.0	71.0	66.0
Rainfall.		3.40	5.10	2.32	5.40	7.31	5.30
Thermometer. Mean.	December.	28.9	38.3	35.7	34.7	25.5	32.6
Thermometer. Range.		46.0	50.5	46.0	61.0	45.0	49.0
Mean barometer.		30.160	30.165	30.176	30.042	30.027	30.114
Mean humidity.		.....	69.9	67.0	76.0	73.0	71.0
Rainfall.		2.74	1.38	2.48	3.37	1.40	2.27
Mean thermometer.	The whole year.	51.8	51.4	52.6	50.3	52.6	51.7
Mean barometer.		30.047	30.041	30.089	30.059	30.037	30.054
Mean humidity.		.....	.....	65.0	67.0	66.0	66.0
Rainfall.		46.79	54.62	46.31	40.24	47.39	47.07
No. of days on which rain fell.		.....	160	136	154	131	142
Prevailing winds.		N. W.	S. W.	S. W.	S. W.	W.	S. W.

and the latter 5° above their respective means. The observation also is interesting that August, notwithstanding its greatly diminished rainfall, reads but one degree below its average, and six degrees above July, in which 5.71 inches of rain fell.

Winds.—The prevailing direction of the wind was, as usual, westerly, but during the months of August, September, and November, it was chiefly from the east. In March dangerous storms were frequent. On July 20 a gale of wind was noted, blowing twenty-four miles to the hour, and on September 17 a disastrous storm occurred, in which the velocity of thirty-four miles to the hour was reached by the wind: the large rainfall of this latter storm has been already alluded to.

Electricity.—The first thunder-storm of the year was recorded in February, and lightning was noted on two occasions in March. Nine storms in July and four in August were accompanied with lightning.

A *lunar eclipse* was observed on March 10, and one of the *sun* on the 25th of the same month.

Epidemics.

The whole number of interments of persons dying in Philadelphia during the year 1876, exclusive of infants prematurely born (244), was eighteen thousand eight hundred and ninety-two (18,892). Assuming the population of the city at the middle of the year to have been 830,000,[1] this gives a ratio of 22.9 deaths to

[1] The basis of this estimate may be found in a foot-note to my report on Meteorology and Epidemics for last year (Trans. Coll. Phys. Philada., 3d series, vol. ii. p. 35. 1876). No account has been taken of the 10,000,000 of "admissions" to the "Centennial Exhibition," as indicating an increase

every thousand of inhabitants living. The mortality was, therefore, somewhat greater than during the previous year, when the ratio was 22.6, and more in excess of the average for the ten preceding years (1866–75), 21.6. This advance in the death-rate is not sufficient to remove Philadelphia from a first rank among the healthiest large cities of the world,[1] but it should be enough to make us more alive to the requirements of sanitary science, since without doubt a more assiduous application of rules of hygiene, already well established, would have spared us this increase.

The deaths were divided unequally between the sexes, the males, as is usual in Philadelphia, furnishing the larger proportion: there were 9848 deaths among them to 9044 among the females.

Within the first year of life, 24 per cent. of the whole mortality occurred; between the first and tenth year 20 per cent.; between the tenth and twentieth 5 per cent.; between the twentieth and fiftieth 28 per cent.; and beyond the fiftieth year 23 per cent. The percentages in the preceding year for the corresponding periods were 23, 24, 5, 25, and 23. The differences are important in a politico-economic view, and tell in favor of 1875, since a life at maturity, at which period the percentage of deaths is greater in 1876, is of more

in our population: for if the daily average of such admissions for six months, 62,000, be distributed through the year, the number dwindles to 31,000, and when it is considered that the far greater number of these was of residents of the city, we reach figures for the strangers small enough to be covered by the possibilities of error in the original estimate.

[1] The annual death-rates per thousand of some other large cities for 1876, were as follows: New York, 27.5; Boston, 24.0; London, 22.3; Paris, 26.6; Berlin, 29.6; Vienna, 27.3; Rome, 28.9 (Annual Summary, Births, Deaths, etc., in London and other large cities, 1876; London, 1877).

TABLE C.—COMPARISON OF CLASSIFIED LISTS OF CAUSES OF DEATH IN PHILADELPHIA DURING 1872, 1873, 1874, 1875, AND 1876.

Class.	Order.	Causes of death.	1872.	1873.	1874.	1875.	1876.
		All causes	18987	15224	15011	17805	18892
		Specified causes	18846	15087	14962	17752	18823
I.		Zymotic diseases	6299	3134	3005	4590	4777
II.		Constitutional diseases	3848	3616	3680	3880	4205
III.		Local diseases	6187	5859	6009	6893	7298
IV.		Developmental diseases	1935	1898	1662	1741	1943
V.		Violent deaths	577	580	606	648	600
		Causes not specified	141	137	49	53	69
			18987	15224	15011	17805	18892
I.	1	Miasmatic diseases	6175	3033	2903	4472	4638
	2	Enthetic diseases	24	33	40	24	34
	3	Dietic diseases	90	57	52	78	87
	4	Parasitic diseases	10	11	10	16	18
II.	1	Diathetic diseases	622	533	469	578	518
	2	Tubercular diseases	3226	3083	3211	3302	3687
III.	1	Dis. of the nervous system	2597	2370	2264	2619	2972
	2	" organs of circulation	776	715	771	835	869
	3	" respiratory organs	1651	1546	1688	1927	1927
	4	" digestive organs	838	922	907	1023	1056
	5	" urinary organs	242	237	273	367	370
	6	" generative organs	35	34	60	57	65
	7	" organs of locomotion	40	31	20	24	24
	8	" integumentary syst.	8	4	26	41	15
IV.	1	Develop. dis. of children	126	124	162	156	154
	2	" " adults	38	45	41	34	37
	3	" " old people	648	513	433	492	599
	4	Diseases of nutrition	1123	1216	1026	1059	1153
V.	1	Accident and negligence	513	521	528	553	518
	3	Homicide	17	14	19	27	22
	4	Suicide	47	45	59	68	60
		Causes not specified	141	137	49	53	69
			18987	15224	15011	17805	18892
I.	1	Smallpox	2580	39	16	56	407
		Measles	140	29	117	12	53
		Scarlatina	168	309	352	1032	328
		Diphtheria	141	106	181	656	708
		Croup	284	188	196	429	386
		Whooping-cough	155	95	74	125	88
		Typhoid fever	341	358	468	400	774
		Typhus fever	35	31	26	21	27
		Erysipelas	76	89	83	87	84
		Puerperal fever	22	28	65	80	54
		Dysentery	75	73	49	78	78
		Diarrhœa	179	163	141	151	149
		Cholera infantum	1603	1068	859	992	1173
		Cholera morbus	111	67	51	47	35
		Malarial fevers	55	30	51	48	47
		Rheumatism	31	45	37	53	53

TABLE C—*continued.*

Class.	Order.	Causes of death.	1872.	1873.	1874.	1875.	1876.
		Cerebro-spinal meningitis	125	238	80	83	85
		Other miasmatic diseases	54	77	57	122	109
	2	Enthetic diseases	24	33	40	24	34
	3	Dietic diseases	90	57	52	78	87
	4	Parasitic diseases	10	11	10	16	18
II.	1	Cancer	334	261	306	319	302
		Other diathetic diseases	280	272	163	259	216
	2	Tabes mesenterica	779	679	721	766	811
		Phthisis pulmonalis	2248	2228	2277	2359	2676
		Other tubercular diseases	199	176	213	177	209
III.	1	Cephalitis	956	874	747	935	1023
		Apoplexy	260	269	232	260	279
		Paralysis	236	233	249	287	305
		Convulsions	732	670	675	811	894
		Sunstroke	135	16	17	20	130
		Other dis. of nervous system	278	308	344	306	341
	2	Pericarditis	45	49	87	87	106
		Aneurism	9	21	18	23	20
		Heart diseases	644	582	626	670	701
		Other dis. organs of circulat'n	78	63	40	55	42
	3	Bronchitis	214	206	256	342	354
		Pleurisy	50	65	82	104	73
		Pneumonia	1252	1132	1133	1277	1306
		Other dis. of respirat'y organs	135	143	217	204	194
	4	Enteritis	306	337	336	399	431
		Peritonitis	131	181	119	159	130
		Intestine diseases	133	111	120	150	133
		Stomach diseases	6	23	37	29	32
		Liver diseases	227	257	227	219	271
		Other dis. of digestive organs	35	13	68	67	59
	5	Nephritis	17	19	24	47	35
		Bright's disease	36	29	131	195	263
		Other dis. of urinary organs	189	189	118	125	72
	6	Diseases of ovaries	6	8	16	13	8
		Uterus diseases	29	26	44	44	57
	7	Caries and necrosis	31	22	10	12	13
		Joint diseases	9	8	10	12	11
	8	Dis. of integumentary system	8	4	26	41	15
IV.	1	Malformations	88	90	123	120	126
		Teething	38	34	39	36	28
	2	Childbirth	10	15	16	10	8
		Puerperal convulsions	28	27	23	24	23
		Other develop. dis. of adults	0	3	2	0	6
	3	Old age	648	513	433	492	599
	4	Debility	822[1]	921[1]	712	695	681
		Inanition	301	295	314	364	472
V.	1	Drowning	99	112	129	119	111
		Otherwise	414	409	399	434	407
	3	Homicide	17	14	19	27	22
	4	Suicide	47	45	59	68	60
		Causes not specified	141	137	49	53	69
			18987	15224	15011	17805	18892

[1] Deaths from "premature birth" are included

value then one in infancy (2–10 years), where the rate of the earlier year is in excess. The variations in the several years are chiefly due to the age-selection of the special zymotics which afflicted them.

The deaths in 1876 were distributed among the different classes of disease, following the classification of Dr. Farr (Table C),[1] thus: Zymotic diseases, 25 per cent.; constitutional, 22 per cent.; local, 39 per cent.; developmental, 10 per cent., and violent deaths, 3 per cent.

Of the 4777 deaths in the first class, 4638 belong in the order of miasmatic diseases, and of this latter number 3776, or more than 80 per cent., fall under six species, namely: Smallpox, 407; scarlatina, 328; diphtheria, 708; croup, 386; typhoid fever, 774; and cholera infantum, 1173. Of these zymotics, typhoid fever, cholera infantum, smallpox, and diphtheria, all show an increase over the preceding year, the advance in the first being specially important, while croup is slightly and scarlatina very greatly on the decrease.

The whole number of deaths from *smallpox* was 407, a long step beyond the 56 of the previous year, and, omitting the great epidemic of 1871–1872, a greater aggregate from this cause than is found in any year since 1865. The pest (Table D), however, at no time showed a disposition to rapid increase or wide extension, the whole number of deaths being counted in a series of small local invasions of the

[1] In this table the deaths from "premature birth" and the number of "stillborn" have been omitted. They were recorded for the several years as follows:—

	1872.	1873.	1874.	1875.	1876.
Premature births .	—	—	229	280	244
Stillborn . . .	825	885	891	824	896

zymotic. For instance, of the 18 deaths which occurred in the first ward, 17 were recorded during the first quarter of the year: these followed upon 3 deaths which took place in the same ward in the last quarter of the previous year: they may all be looked upon as belonging together, and as being a part or offspring of the small epidemic which prevailed in the adjacent second and twenty-sixth wards in the latter half of 1875. The same may be said of the 6 deaths from variola which took place in the contiguous fourth ward during the first six months of last year. The municipal divisions just spoken of are all included in a group referred to in my last report as being specially the seat of zymotic disease, a locality which was severely ravaged by the epidemic of 1871–72.[1]

In the wide expanse of territory to the north of this group, known formerly as the "city proper" (fifth, sixth, seventh, eighth, ninth, and tenth wards), there were only 8 deaths from smallpox, all taking place before the month of May, and of the deaths recorded for these two districts together, in all 49, only 8 happened in the last half of the year. On the other hand, in the group formed by the seventeenth, eighteenth, nineteenth, and thirty-first wards the deaths, though extending through the whole year, were more numerous in the later months. This is the other district referred to in my last report, as appearing generally more susceptible to zymotic disease, and it was also visited severely by the epidemic of 1871–72. Much the larger proportion of deaths from smallpox last year occurred in this and adjacent parts of the city:

[1] Report of Board of Health of City and Port of Philadelphia to the Mayor for the year 1872, pp. 60–61. Philadelphia, 1873.

TABLE D.—DISTRIBUTION OF THE DEATHS FROM THE ZYMOTIC DISEASES SMALLPOX, SCARLATINA, DIPHTHERIA, CROUP, AND TYPHOID FEVER, AMONG THE SEVERAL WARDS OF PHILADELPHIA IN 1875, DURING EACH MONTH AND FOR THE YEAR.

Number of ward.	January.					February.					March.					April.					May.					June.					July.					August.				
	Smallpox.	Scarlatina.	Diphtheria.	Croup.	Typh'd fev.	Smallpox.	Scarlatina.	Diphtheria.	Croup.	Typh'd fev.	Smallpox.	Scarlatina.	Diphtheria.	Croup.	Typh'd fev.	Smallpox.	Scarlatina.	Diphtheria.	Croup.	Typh'd fev.	Smallpox.	Scarlatina.	Diphtheria.	Croup.	Typh'd fev.	Smallpox.	Scarlatina.	Diphtheria.	Croup.	Typh'd fev.	Smallpox.	Scarlatina.	Diphtheria.	Croup.	Typh'd fev.	Smallpox.	Scarlatina.	Diphtheria.	Croup.	Typh'd fev.
1st	6	2	2	5	2	8	..	3	3	1	3	1	3	2	3	..	1	3	2	1	..	3	..	..	1	..	..	..	1	2	..	2	..	1	8	1	3	4	1	3
2d	1	1	..	1	1	1	1	..	3	2	..	3	..	..	..	..	..	2	1	..	..	2	2	2	2	..	2	..	2	2	1	3	..	2	1	..	1	1	..	..	1	3
3d	..	1	2	1	..	..	..	1	1	..	1	2	1	2	..	1	1	6	1	2	..	..	..	..	..	..	..	..	1	1	..	..	..	..	..	..	..	1	1	1
4th	..	..	2	..	1	1	1	1	3	1	2	..	1	2	1	2	..	2	1	..	1	..	..	..	..	1	1	..	1	..	..	..	2	1	..	..	..	..	3	1	3
5th	2	1	1	..	2	..	..	..	..	1	..	1	..	1	..	..	..	..	..	..	..	..	1	..	..	2	..	2	2	..	2	..	1	..	..	..	..	..	..	..
6th	..	1	..	..	..	..	1	..	..	..	..	2	..	1	2	..	1	..	..	..	1	..	2	2	1	..	..	..	..	1	1	..	..	..	..	..	..	..	..	2
7th	1	..	2	1	1	..	..	3	2	1	2	..	3	1	1	..	..	1	..	..	..	2	2	2	..	..	..	1	3	..	1	..	..	1	1	4	..	..	1	1	11
8th	..	..	4	..	..	1	1	1	1	1	..	..	1	1	1	2	1	1	..	..	3	..	2	1	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	6
9th	..	1	1	..	..	..	..	1	..	..	..	3	..	..	1	..	1	..	..	..	..	..	..	..	..	2	..	1	..	..	..	..	..	..	..	..	..	..	..	..	3
10th	..	1	..	1	1	..	2	..	2	1	..	2	3	..	..	..	..	..	..	..	..	..	..	1	..	..	..	6	..	2	..	..	1	..	5	..	..	..	..	..	3
11th	..	..	2	..	1	..	1	..	1	..	..	1	..	1	2	..	..	2	1	..	6	..	..	3	..	..	1	..	2	..	..	..	1	..	..	..	..	..	..	..	2
12th	..	1	1	2	1	..	..	1	..	1	..	2	2	..	..	..	..	2	1	..	1	1	3	..	2	1	..	1	..	1	1	..	..	..	1	..	..	1	1	2
13th	..	2	3	..	1	..	..	2	1	1	..	..	7	2	..	..	..	1	2	..	..	..	2	1	1	..	..	4	1	1	..	..	..	..	..	..	..	2	..	1
14th	2	..	5	2	2	..	..	2	4	2	..	..	3	3	..	3	1	2	1	2	9	1	6	1	1	5	..	2	1	2	3	1	..	..	4	..	..	..	..	2
15th	..	8	9	5	3	2	2	12	4	2	5	6	5	6	1	7	3	6	..	1	2	3	6	3	4	4	..	3	5	..	3	1	..	..	2	..	..	1	..	3
16th	..	2	5	..	1	..	3	2	2	..	1	1	..	..	3	..	1	..	2	..	3	2	..	1	1	2	..	..	1	4	1	..	..	..	2	1	..	..	..	3
17th	..	2	..	..	2	1	1	..	4	..	..	..	3	2	3	1	..	2	1	1	1	..	1	2	1	..	..	4	1	3	1	..	1	..	1	..	..	..	..	4
18th	..	3	3	1	3	..	2	1	..	5	1	6	1	1	4	1	5	..	1	5	..	1	5	1	3	2	..	1	..	2	..	..	..	..	3	..	..	..	..	3
19th	6	2	5	3	8	2	3	3	5	3	4	4	5	2	1	6	2	1	6	2	11	3	5	2	4	9	..	2	2	3	11	1	..	..	7	15	..	..	3	9
20th	1	1	..	3	3	2	..	2	2	2	1	3	3	..	3	..	1	5	..	2	1	2	4	2	1	..	..	6	..	1	..	1	1	..	6	1	..	4	1	5
21st	..	7	6	5	..	..	4	3	5	1	..	8	4	3	1	1	2	4	2	..	..	2	1	3	..	..	2	3	..	..	..	3	3	1	1	..	2	1	..	..
22d	..	3	2	..	1	..	1	1	..	2	3	3	6	..	..	3	..	5	..	1	1	..	6	..	1	..	..	..	1	..	2	..	..	1	1	..	..	..	..	3
23d	..	..	1	..	2	..	3	2	1	1	..	..	3	3	1	2	..	1	1	3	1	..	4	1	..	..	..	1	2	1	..	..	2	..	2	..	..	1	..	2
24th	..	3	6	2	1	..	1	..	2	2	..	7	7	..	1	..	1	2	..	..	..	2	..	3	..	3	3	3	..	2	..	..	1	1	3	..	..	1	2	10
25th	2	5	4	5	2	..	1	3	3	1	2	2	5	1	3	..	8	3	2	..	2	2	..	3	1	1	1	9	1	2	2	..	4	1	1	..	..	2	2	1
26th	..	2	3	2	1	1	1	1	..	..	..	1	1	2	..	1	1	2	..	1	..	2	2	2	1	..	1	1	..	2	..	..	1	..	4	..	..	2	..	5
27th	..	..	1	..	2	2	1	2	..	3	..	..	..	1	..	..	..	1	..	..	3	..	3	1	..	3	1	3	..	3	..	1	..	..	4	..	..	..	..	5
28th	1	1	2	2	..	12	..	4	3	1	17	..	3	1	1	4	1	3	2	..	10	3	2	2	2	11	..	1	1	..	..	2	..	..	..	..	1	..	..	2
29th	1	1	3	..	3	..	2	..	2	..	1	1	6	1	4	4	2	1	1	..	2	2	3	1	3	1	1	5	..	4	1	..	1	1	..	1	..	6	..	10
30th	..	1	3	1	..	..	..	2	2	..	..	2	3	1	..	1	2	2	2	..	..	..	1	..	1	1	1	..	..	..	..	1	2	2	..	..	1	..	6
31st	..	1	4	..	2	..	3	3	1	1	4	6	1	2	7	4	2	2	1	..	3	1	1	..	4	4	2	2	2	1	1	1	..	2	2	..	..	2	..	3
Total.	26	55	82	42	47	33	35	56	57	37	47	66	81	42	43	45	38	57	29	36	49	38	73	32	43	47	20	62	23	44	28	17	24	8	67	19	6	32	17	116

TABLE D—*continued.*

Number of ward.	September.					October.					November.					December.					For the year.					Population, Municipal census of 1876.	Area in square miles.
	Smallpox.	Scarlatina.	Diphtheria.	Croup.	Typh'd fev.	Smallpox.	Scarlatina.	Diphtheria.	Croup.	Typh'd fev.	Smallpox.	Scarlatina.	Diphtheria.	Croup.	Typh'd fev.	Smallpox.	Scarlatina.	Diphtheria.	Croup.	Typh'd fev.	Smallpox.	Scarlatina.	Diphtheria.	Croup.	Typh'd fev.		
1st	..	..	4	4	2	..	..	5	4	2	..	..	6	1	4	..	..	2	3	3	18	12	31	27	32	38,794	5.509
2d	1	1	3	..	4	..	1	1	4	1	..	3	1	2	3	..	..	2	1	3	5	15	12	15	25	28,242	.442
3d	..	..	1	..	1	..	..	1	1	2	..	..	..	2	1	1	..	2	2	..	3	4	15	12	8	20,255	.191
4th	..	..	4	1	3	..	1	4	2	..	..	..	..	1	2	1	..	1	..	2	8	4	20	11	14	20,545	.229
5th	..	..	..	..	2	..	..	2	..	3	..	..	..	..	1	..	1	1	1	1	2	5	8	2	14	18,972	.321
6th	..	..	..	..	..	..	..	1	..	..	..	..	..	..	1	..	..	..	..	2	..	7	3	3	9	12,670	.321
7th	..	..	2	..	2	..	1	4	..	2	..	..	2	1	2	..	1	5	1	2	3	5	29	8	27	33,067	.439
8th	..	..	..	..	2	..	1	..	..	..	..	..	..	..	5	..	..	2	..	4	3	5	10	3	22	23,868	.435
9th	..	..	..	..	4	..	..	..	..	1	..	..	..	..	..	..	..	..	..	4	..	6	2	..	15	15,915	.400
10th	..	..	1	1	6	..	..	2	..	2	..	..	..	..	3	..	..	1	1	4	..	5	14	6	27	24,786	.359
11th	..	..	..	..	3	..	..	1	..	4	2	..	..	..	..	..	..	5	..	1	9	1	15	4	15	14,345	.210
12th	..	..	..	..	1	1	..	2	..	..	2	..	..	1	..	..	..	..	..	3	6	4	13	5	12	15,394	.193
13th	..	..	..	..	1	..	..	1	..	4	..	..	..	..	1	..	..	..	..	3	..	4	22	9	13	20,027	.259
14th	..	..	1	..	4	..	..	1	..	1	..	..	..	1	2	2	..	4	2	7	24	3	26	15	29	23,385	.237
15th	1	1	2	2	6	..	..	9	3	6	..	..	3	4	8	..	..	2	..	10	24	24	58	32	46	48,472	1.049
16th	1	..	..	3	2	1	..	2	..	1	..	2	1	2	1	2	1	..	1	..	12	12	10	12	18	18,903	.281
17th	2	..	1	..	2	1	1	4	2	3	..	..	2	4	..	3	..	2	3	..	10	4	20	19	20	21,279	.251
18th	..	..	2	1	5	..	1	2	4	2	..	..	1	2	3	2	..	..	..	3	6	20	16	11	41	28,286	.650
19th	5	1	..	2	4	15	2	2	3	4	15	1	4	3	6	7	..	2	4	3	106	19	29	35	54	40,604	.742
20th	..	..	2	..	7	..	1	2	2	3	3	1	7	3	5	3	1	4	2	9	12	11	40	15	47	41,854	.734
21st	..	1	4	..	4	..	4	4	1	1	1	4	4	1	..	..	3	2	2	..	2	42	39	23	8	18,097	7.129
22d	..	..	5	..	1	..	1	11	..	3	..	..	..	2	2	..	1	7	1	3	9	9	43	5	18	28,482	18.114
23d	..	..	..	..	3	..	..	3	2	2	1	..	5	1	..	..	..	4	2	3	4	3	27	13	20	25,299	42.716
24th	..	..	8	3	4	..	..	4	1	6	1	2	1	2	2	1	2	8	1	6	4	19	44	14	40	41,310	9.725
25th	..	..	2	2	1	3	1	3	5	..	2	..	..	2	2	1	1	3	2	2	15	19	41	27	18	28,648	13.660
26th	1	..	..	1	2	..	1	1	1	2	1	..	..	..	2	..	..	..	..	2	4	9	14	8	22	27,905	7.481
27th	..	..	..	..	2	..	1	..	..	2	..	1	..	..	1	..	..	1	..	2	3	10	6	1	30	22,457	11.680
28th	2	1	1	2	1	5	..	3	3	3	8	1	3	2	..	2	2	3	..	2	75[1]	11	26	21	12	24,381	6.343
29th	..	..	3	1	15	..	..	6	2	4	..	..	1	..	5	..	..	1	..	12	13	10	37	9	61	33,974	1.460
30th	..	..	..	..	2	1	..	..	..	3	..	..	..	1	3	..	2	3	1	5	3	10	16	7	21	28,937	.519
31st	3	..	1	1	..	1	..	2	2	1	5	..	2	1	2	3	1	1	1	6	24	16	22	14	33	28,895	.713
Total.	16	5	47	24	96	28	17	83	42	68	41	15	43	39	67	28	16	68	31	107	407	328	708	386	771[2]	817,448	129.579

[1] The hospital for smallpox patients is situated in the 28th ward.
[2] The whole number of deaths was 774, but one in July and two in December were unrecorded as regarded locality.

especially did the nineteenth ward suffer, and the thirty-first, which till recently formed a part of the nineteenth. In the former ward the deaths were in all 106, scattered through every month in the year: beginning at 6 in January, they increased slowly and irregularly, reaching maxima of 15 in August, October, and November, and then falling to 7 in December. The fact that the localities chiefly affected in the small epidemics are the same that were ravaged severely in 1871–72, illustrates the infectious nature of the variolous poison in contradistinction to its purely contagious properties, and emphasizes the analogies of smallpox with other diseases of its order.

The decline of the late epidemic of *scarlet fever*, which was already evident in the latter months of 1875, was continued through 1876, the whole number of deaths from the zymotic in the latter year being but two-thirds of those in the former, and only one-fourth occurring in the closing six months. The number of deaths fell off in all the wards except the outlying twenty-first, which contains the town of Manayunk: here, where the mortality had not been especially severe the year before from scarlatina, though in excess from diphtheria and croup, the deaths were much more than doubled, the aggregate equalling in proportion those in the most severely scourged wards during 1875. The greater mortality, however, belonged to the earlier months.

The apprehension excited by the increasing number of deaths from *diphtheria* towards the end of 1875, proved to be not without foundation, since in 1876 the mortality from this disease was augmented 8 per cent. The deaths reaching the high figure of 708,

though distributed throughout the whole city, were especially numerous in the northwestern sections, and in the outlying towns of Manayunk, Germantown, and Frankford. I called attention last year to the virulence of diphtheria in Manayunk in 1875, and I found that town to be still in 1876 far ahead of the other sections of the city in its mortality from this disease, though the number of deaths had fallen off as compared with 1875 one-half. The deaths throughout the city were most numerous in the first quarter, 219; there was a falling off in the second to 192, and again in the third to 103, but a rise in the last quarter to 194. This relation to the seasons is in accordance with the statistics of Wibmer;[1] I have brought it out more clearly by aggregating the deaths in the several quarters, since arranging the months in the order of their excess in mortality they read rather confusedly as follows: October, 83 deaths; January, 82; March, 81; May, 73; December, 68; June, 62; April, 57; February, 56; September, 47; November, 43; August, 32, July, 24. The diminution of the number of deaths in the last quarter from that in the first, and from that in the corresponding quarter of the previous year, allows the indulgence of the hope that the epidemic which began in the fall of 1874 was on the wane in 1876; but the experience of the course of diphtheria in Philadelphia in the past forbids the expectation of a rapid decline.

The deaths from *croup* were scattered over the city in pretty much the same localities as in the previous year, and the decrease of 10 per cent. (43) was divided among nearly all the municipal divisions. Certain

[1] Quoted by Oertel, Ziemssen's Cyclop. Pract. Med., Art. Diphtheria, vol. i. p. 583, Amer. edit.

wards, however, the fourteenth, fifteenth, and twenty-eighth, show a doubled mortality. The ward which suffered most was the twenty-first (Manayunk), as was the case also in the previous year, though in 1876 there was a decrease of one-third the number of deaths as compared with 1875. I have alluded above to the exaggerated sway of diphtheria in the same ward in the corresponding periods. This parallelism of the two diseases was observed in some other sections of the city, and would appear very significant were it not that its absence is equally conspicuous in some other wards, especially in the seventh, tenth, eleventh, twentieth, twenty-second, twenty-fourth, and twenty-ninth. The order of the months according to their degree of mortality is much more regular with respect to the several seasons than that which is recorded above for diphtheria: it is February, 57 deaths; January, March, and October, each 42; November, 39; May, 32; December, 31; April, 29; September, 24; June, 23; August, 17; July, 8.

It need scarcely be said that the deaths from *cholera infantum* were more than ordinarily numerous in a summer the July of which was distinguished by a continued intensity of heat without precedent. Yet it is worth notice that there have been years in which the number has been much exceeded, as, for instance, 1872, when it was surpassed more than 40 per cent., and 1873, when in proportion to population it was 5 per cent. greater; in both these years unusually high temperature prevailed, the summer of 1872 having been the hottest on record. The lower rate last year coincided with an August cooler than that of 1872, and with somewhat less humidity of the atmosphere in that

month than in the corresponding period in the other years. It is difficult to trace any connection between indifferent local hygienic conditions and the prevalence of cholera infantum; the wealthy eighth ward was not more exempt than the less favored nineteenth. Moral degradation of parents, through the neglect of offspring which it involves, is an efficient ally to this slayer of infants; witness the ravages of the disease among the miserable inhabitants of the fourth ward.

The most striking feature of the bills of mortality for last year is the increase in the number of deaths from *typhoid fever*. The mortality from this cause is 774, nearly double that for the previous year, and has not been approached in height since 1865, when there were 773 interments of those dead from the disease, in a population one-third less. After that year, for some reasons an exceptional one, the annual numbers of deaths from the fever were diminished one-half, and have little exceeded that proportion in any year until the last.

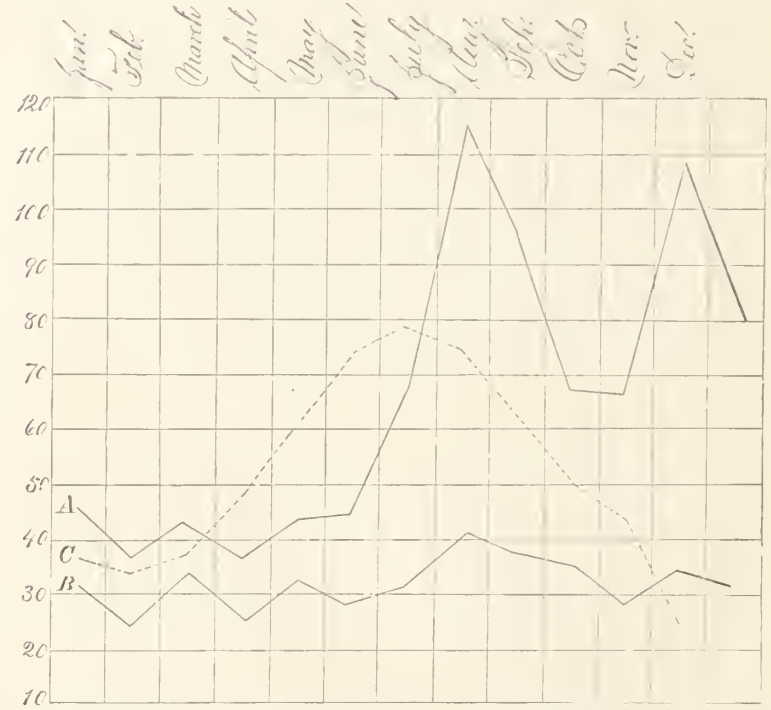
Of the 774 persons who died from typhoid fever in 1876, the ages of 766 have been recorded, as follows: 134 or 17.5 per cent. were under 15 years of age; 375 or 49 per cent. between 15 and 30 years; and beyond 30 years, 257, or 33.5 per cent.; the mortality being greatest, as is usual from this fever, in the periods of youth and early manhood.

More than two-thirds of all the deaths took place in the latter half of the year, August being distinguished among the months by the most, and next December. This is shown in the highest of the curves in the accompanying figure (Fig. 1, *A*) constructed to represent the monthly fluctuations of the deaths from

this disease during 1876. The lowest curve, *B*, in the same cut, expresses the corresponding differences in the averages for the several months over a period of ten years (1866–75). These two crooked lines are in remarkable coincidence in their ups and downs, though

Fig. 1.

Curves representing the monthly mortality of typhoid fever during 1876, the average monthly mortality of the same for ten years (1866–1875), and the mean monthly temperature for 1876.



A. Mortality curve for 1876. *B*. Mortality curve of averages. *C*. Curve of temperature.

in different planes, until the month of June is reached, when the curve for 1876 begins to rise a month in advance of the curve of averages; after June they

both ascend, each attaining its greatest height in August, but that of last year rising immensely more than the other; then they both fall till minima in November are reached, the curve of averages being now as low as when its greatest rise began, while the other is still very high above its May point: once more both curves ascend, making peaks in December, but that for 1876 is again greatly exaggerated. Between the mortality curves, I have introduced one of temperature, *C*, which is seen to be in general accord with the other lines in respect to their rise to the August maxima and their subsequent decline, except that its apex is reached a month earlier. This relation of typhoid fever to season, its "periodical disposition" (Liebermeister), has been referred to by many writers on the disease.

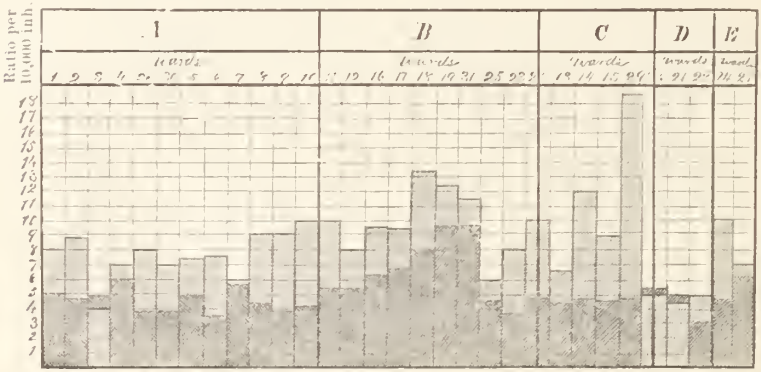
The increase of deaths from typhoid fever last year appears to be distributed over nearly every ward of the city, when the mortality of each is compared with its respective average from this cause for ten years (1866–75), (Fig. 2): but the excess is, as a rule, 100 per cent. in those municipal divisions where the average number of deaths is smaller, and 50 per cent. in those where the greater mortality usually exists. The chief exceptions to this general statement are the twenty-first ward (Manaynnk), and the twenty-eighth, a rural ward, each of which is a little behind in its number of deaths; and the twenty-ninth (formerly part of the twentieth), in which the mortality is increased almost 400 per cent.

To estimate the comparative mortality of the epidemic, I have had recourse to the records of the principal hospitals of the city, which have been kindly

placed at my service. I find that of 254 patients, suffering from typhoid fever, who were admitted into these houses, 53 died, or one in every 4.8: which is

FIG. 2.

A representation of the mortality from typhoid fever in the several wards of Philadelphia during the year 1876, and of the average annual mortality of these municipal divisions from the same cause during a period of ten years (1866–1875). The lighter columns indicate the former, the darker the latter. The wards are arranged with reference to their sources of water supply, which are designated at the top of the diagram. The figures to the left express the number of deaths per 10,000 inhabitants from the fever. In computing the ratios for 1876, the municipal census taken in April of that year has been used; for those of the averages, the United States census of 1870.



A. Schuylkill water from Fairmount reservoir. *B*. Delaware water from Kensington reservoir. *C*. Schuylkill water from Spring Garden reservoir. *D*. Partly Schuylkill water from Roxborough reservoir. *E*. Schuylkill water from Belmont reservoir.

not a very different ratio from that given by Murchison, as obtaining in the London Fever Hospital, during a period of fourteen and a half years—1 in 5.38.[1] It must not be forgotten, however, that the cases received into hospital are the most serious ones, so that this ratio will be too high if applied to the whole

[1] Treatise on Continued Fevers of Great Britain, by Charles Murchison, M.D., London, 1862, p. 528.

number of cases which occurred in the city. With this understanding the data are sufficient to warrant the assertion that the epidemic was one of moderate severity. I am not aware of any special features predominating in the clinical history of the cases: those which I myself saw in hospital and private practice presented different varieties of the disease, the comparative mortality being greatest in the summer. Of seven cases under my care in private practice during August, when the disease was at its height, one proved fatal: this was in a youth of 19; the course of the disease was typical, except that the head symptoms were more prominent than usual: death took place at the end of the third week from exhaustion.

Looking into the causation of the epidemic, no factor suggests itself with such force as the unusually high temperature of the year, and especially the unwonted heat of the summer. "It would seem," says Murchison, speaking generally of typhoid fever, "that the cause of the disease is only exaggerated or called into action by the *protracted* heat of summer and autumn, and that it requires the *protracted* cold of winter and spring to impair its activity or to destroy it."[1] The same author refers besides to the unusual prevalence of the disease after exceptionally hot summers in Great Britain, and gives the authority of Cless in Germany, and De Claubry in France, for a similar sequence in their respective countries.[2] Dr. Ernest Besnier, also, writing recently of the epidemic which was prevailing in Paris last summer and fall, calls attention to the same relation.[3] Modern theories

[1] Op. cit. p. 416. [2] Ibid. pp. 419–420.

[3] L'Union Médicale, Nov. 4, p. 667, et Nov. 7, p. 679.

concerning the etiology of typhoid fever explain very readily the agency of heat in fostering the disease: for the products of putrefaction, on which its special poison is supposed to feed, must be in greater quantity when the sun is developing more power in climates where there is always a certain amount of moisture in the air. But the extraordinary heat of last summer was coincident with another exceptional meteorological condition, a very small rainfall in August: from this, a lowering of the water in the soil would follow, a circumstance which Buhl[1] has shown to be accompanied with an increase in the prevalence of typhoid fever, whatever the explanation of the fact may be.

We had then atmospheric and telluric conditions last year favorable to an epidemic of the fever. And if we accept the theory of the spontaneous generation of the special poison from putrefying substances, we have gone far enough in explaining its exaggerated production. But should it be maintained, with Budd,[2] that the poison is always derived from the alvine evacuations of a person suffering with the disease, we must take a step further. It would be sufficient to reply that typhoid fever with its poison is always endemic here; but can we not surmise, to explain a certain number of the cases, a fresh importation of the poison as well? Is it being too speculative to imagine that of the hundreds of thousands of strangers who visited our World's Fair last year, there were many who brought with them to a conge-

[1] Quoted by Liebermeister. Ziemssen's Cyclop. Pract. Med., Art. "Typhoid Fever." Am. ed p. 68.

[2] Lancet, 1856, vol. ii., and 1859, vol. ii.

nial soil the germs of this disease? Such an idea would receive some confirmation from an analogous condition as regards the influx of people in the year 1865, which has been referred to above as having been remarkable for an even severer epidemic of typhoid.

In that year, the last of the civil war, the numerous large hospitals in Philadelphia were being constantly filled up with invalided soldiers from the south, while thousands of troops were arriving to be mustered out of the military service. Fever poison thus introduced from without, might be expected to be more virulent among the inhabitants of the city to which it was brought, as a corollary to the accepted proposition[1] that new-comers in an infected locality are more prone than residents to be attacked by the disease. Moreover, the President of the Epidemiological Society of London, in his recent annual address,[2] quotes with approval the explanation of Dr. Léon Colin, with reference to the outbreak of smallpox in Paris in 1870, that the virus of variola, brought there with the soldiers from Brittany, was more malignant than that already existing in the Capital, and thereby responsible for that epidemic. If such varying virulence may be assumed for the poison of smallpox, why not also for that of typhoid fever?

Given the poison, through what medium and whence did it enter the systems of those affected with the disease? To these questions I shall only attempt general answers, since I have had neither the opportunity nor the time to investigate, as would be

[1] Murchison quotes several authorities with reference to the disastrous effect of recent residence in an infected locality. Op. cit., p. 425.

[2] Practitioner, vol. xvii., 1876, p. 468.

necessary for a more specific reply, each of the local outbreaks of the disease. For this reason I will overlook such supposed agencies as *infected milk* and individual instances of the entrance of sewer gases into houses, accidents which are necessarily limited in their effects.

As soon as the weekly returns of deaths in the city for August showed an alarming increase in those from typhoid fever, an onslaught upon the character of the water-supply was made by the press of neighboring cities, which found an echo even in that of Europe. This illustrates the great importance which is attached to drinking-water as a vehicle of the poison: and the fact that Philadelphia is furnished with river-water, into which a certain amount of *sewage* is received, made the attack appear not unreasonable. Yet, on examination, I think that the charge will not be sustained. In Fig. 2, columns in different degrees of shading have been drawn; the darker to represent the average annual mortality during ten years (1866-75) from typhoid fever in the several wards of the city, and the lighter their respective mortality for last year. These columns are arranged according to the contiguity of the wards and their respective sources of water-supply, these sources being noted for the several groups. Of the four reservoirs supplying the principal divisions, three contain water taken from various points in the Schuylkill River, and one is filled from the Delaware.

The order of purity of these basins, as regards freedom from sewage is, first, the Fairmount, then the Belmont, next the Spring Garden (these supplied from the Schuylkill), and, most polluted of all, that fed

from the Delaware River, the Kensington. In a city poisoned by infected drinking-water, so universal would be the direct exposure of the inhabitants to the poison that I should expect the deaths from its effects to be distributed quite evenly over sections furnished with the same water-supply, and, where this was different, the degree to which the several sources were polluted with organic matter might be looked for to establish a difference. A glance at Fig. 2, however, will discover a great irregularity in the height of the lighter columns (representing the mortality of 1876) in the several groups, and in their respective increase or diminution over the darker columns indicating the average mortality. The increase, moreover, is not greater in that group which has the most impure water-supply, nor is the increase in the several wards of individual groups proportioned by that of the section in which they are situated. For instance, the twenty-ninth ward, which far surpasses any other in its mortality last year, and in its increase over its average, belongs in the group furnished from the Spring Garden reservoir, which stands before the Kensington in respect to the purity of its water: at the same time the thirteenth ward, in the same section, records fewer deaths and a less increase than many others in the Fairmount group with the least sewage-polluted water.

To pursue this subject further, I took the trouble to mark out on a map of the streets of the city the residences of those who died from typhoid fever last year. I obtained such results as follow. Having divided the fifteenth ward into four sections, each a little less than a half mile square, it appeared that in

one of these divisions a single death had occurred; in a second, ten; in a third, fifteen; and in a fourth, eighteen. Within a circumference having a radius of four hundred feet, with the intersection of Second Street and Girard Avenue for a centre, ten deaths took place; from within a circle of equal size, drawn about the crossing of Fourth Street and Girard Avenue, but five deaths were returned; and a circle could be made tangent to the first one, of twice its dimensions, in which not a single death occurred. In the fourth ward, of fourteen deaths which were reported from there, six occurred in a block of less than four hundred feet square, and six more in a locality of about equal size, two squares to the east: while nearly half of all the deaths in the territory of the second, third, and fourth wards combined, took place in an area of fifteen adjacent squares, the whole space covered by these municipal divisions equalling one hundred and fourteen such units of measure.

This uneven distribution through the city of deaths then opposes the idea that poisoned water was responsible for any considerable part of the increased mortality from typhoid fever. Air contamination remains. This may arise from a mingling of the exhalations from the sewers with the atmosphere, an event considered especially obnoxious with reference to the development of typhoid fever. The general distribution of these subterranean passages under the city renders it possible to explain through their agency a very wide extension of the disease. But against the use of these, to elucidate the greater prevalence of the disease in Philadelphia last year, two facts are especially prominent: the first, that a

certain concentration of these gases is necessary to develop their poisonous properties, as, for instance, confinement in the rooms of a house, since in towns where ventilation of the sewers is practised, no detriment has been observed to follow their escape into the streets; and, second, that the houses here, as a rule, do not have direct communication with the sewers, the chief functions of which are to carry off the overflow from rain storms and the contents of the kitchen waste-pipes, which sometimes run a long distance exposed to the air before the sewer-inlet is reached.

These considerations seem to me to give a denial to any extended effect of sewer emanations in aggravating our mortality from typhoid fever; but it may be further negatived by means of certain data obtained from the Highway Department.[1] From these it appears that sewer connections (of water-closets, stationary washstands, and house drains) are more numerous in the eighth, ninth, and tenth wards than in some other portions of the city, as, for instance, in the sixteenth, seventeenth, eighteenth, and nineteenth wards: yet in the latter the mortality from typhoid fever exceeded that of the former, last year, and the same is true as regards the averages for ten years. Further, one group of six deaths which was spoken of above, as occurring in a very limited space in the fourth ward, was found at an "apex" of territory unprovided with sewers, these taking their origin all about it, and a locality in the nineteenth ward, near the intersection of Front and Cumberland

[1] Oral information.

Streets, where another group of deaths took place, was equally unprovided with these large drains.

A more probable source of pollution to the air, in my estimation, and one sufficiently extended to explain so general an epidemic, is the cesspool system of disposal of alvine excreta which prevails in Philadelphia. The vault, which this requires, occupies a place in the rear of every house, except that the meanest tenements are sometimes clustered round one which does duty to the whole group. These receptacles are often very deep, and their contents frequently undisturbed till their level approaches that of the surrounding surface. Directly from these the foul air carrying the poison ascends to reach the persons of those who visit the contracted houses which cover them: or it may even enter the dwelling-house, the doors and windows of which sometimes are but a few feet distant from the outside office. Again, the pits are often but imperfectly lined, allowing the noxious matters to penetrate into the surrounding soil: whether the poison can thence undisturbed reach the atmosphere above, may be doubtful; but as our streets are constantly being excavated for laying down or repairing gas and water pipes, and the yards of the houses and adjacent lots dug out for new buildings, the infected soil is likely to be at some time exposed directly to the upper air.

I will recur to the curve of mortality for 1876 (Fig. 1, *A*) to point out its rapid fall from August to September, and its subsequent great ascent in December. Since the first took place spontaneously, so to speak, that is, without interference on our part, it should warn us not to conclude too quickly in these

eases, where a local epidemic subsides after some particular measures have been taken to oppose it, that these have effectively prevailed against it. With reference to the ascent, how shall it be explained? Not by the explosion of a secondary *local* epidemic, for the increase of mortality was distributed over a wide extent of territory: but probably by the hypothesis of a continuous succession of "crops" of the poison, the duration of vitality of each being governed by not well understood laws. Some such view as the latter seems necessary to understand the regularity with which the peaks in the curve of 1877 accord with those in the curve of averages.

The above remarks are consistent with the theory that makes typhoid fever dependent on the action of a special poison: this is found in the excreta, and is capable of propagation in decomposing substances; "it travels from the diseased individual to the localities which are favorable to its growth and multiplication, and from thence again into the human body."[1] If the theory is true, and the laws of causation have been rightly applied above, the disease in Philadelphia might be successfully combated by some new method of disposing of the alvine excreta; as, for instance, a more complete and efficient system of sewerage. In some cases the poison might be attacked in a more direct manner. Since it is believed that this may be destroyed by a heat approaching that of boiling water (212° F.), and by the action of some disinfectants, let the stools from fever patients be received into a vessel in which there has been placed an antiseptic solution of, for instance, carbolic acid,

[1] Liebermeister, op. cit., vol. i. p. 52.

or—the smell of this being disagreeable—of chloride of zinc: this would destroy also any chance of offensive odor, and, as a further precaution, the mass might afterward be transferred to another vessel containing boiling water.

In analyzing the causes of death, and the classes of disease other than the zymotic, I have attempted to discern any disturbing influence in the death-rate from the great social event of last year, our International Exhibition. I have been unsuccessful, unless I am right in attributing some excess in the deaths from "phthisis pulmonalis," and from "old age," during the spring and fall, to the accelerating effect of the fatigue induced by the examination of its marvels and its beauties. The number of persons of advanced age who visited the Exhibition was remarkable, and greater than could be explained by the curiosity and interest naturally excited by the show itself: these were supplemented by the nature of the occasion seized upon for the event, the commemoration of the Centennial birthday of the Republic, and it was this feature which seemed to be especially attractive to those who had lived longest under our benign government. The deaths from "phthisis pulmonalis" were greater last year than ever before, and the general order of the mortality as regards the seasons was disturbed, the autumn, in which, as a rule, the least number of deaths occur, coming next to the spring, which, as usual, was distinguished by the most. It is difficult, with a disease which may run so chronic a course as consumption of the lungs, to weigh the influence of a special season upon its

death-rate. Yet it is significant that the increased mortality coincided with a cold September, and an autumn characterized by a heavy rainfall and an unusual prevalence of easterly winds.

The six per cent. increase of deaths over the previous year in the class of Local Diseases, may be traced to the agency of the excessive heat of the summer; it was manifested particularly in an augmented mortality in the order of diseases of the nervous system. The excess here fell chiefly among children, and was due to the occurrence of more cases of cephalitis and convulsions; but adults suffered also from sunstroke. There was the large number of 130 deaths from the latter cause, while but 30 were recorded from the same disease in the previous year. Among the diseases of the digestive organs there was but a small increase, notwithstanding the high summer temperature.

The greater mortality of 1876, in the class of Developmental Diseases, over what occurred in 1875, fell upon the very old and the very young alike, reaching the latter through inanition. For both classes of persons the autumn especially was more fatal. With respect to the old, a partial disturbing cause has been alluded to above, but to these, as well as to the infants who had barely survived the heats of the summer, the wet fall was to be looked upon as unfavorable.

It must be a matter of surprise to those who saw the scrambling crowds surging among the vehicles about the gates of the Exhibition, crowding the platforms, and literally hanging on to the sides of the

over-filled horse and steam cars, that the number of killed noted in the class of Violent Deaths should be less than that in the previous year, yet such is the gratifying record. An instance is furnished of the efficiency of the safeguards which our enlightened age places about the more obvious dangers to human life.

HEMIOPIA AND DECUSSATION IN THE OPTIC CHIASM.

By

GEORGE C. HARLAN, M.D.,

SURGEON TO WILLS HOSPITAL, OPHTHALMIC AND AURAL SURGEON TO THE CHILDREN'S HOSPITAL.

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The theory of the semi-decussation of the fibres of the optic nerves in the chiasm, is often spoken of as Wollaston's Theory. Wollaston is generally supposed to have been the first to suggest it, in 1824, to facilitate the explanation of a transient hemiopia occurring in his own case; but it is known that Sir Isaac Newton advocated the possibility of a semi-decussation in 1704. The name of Müller is also associated with this theory, and it has been adopted, without question, in nearly all the standard text-books on anatomy, physiology, and ophthalmic surgery.

Within a few years, this long rest has been broken by the defection of a number of pathologists and physiologists, who have advocated the theory of total decussation, maintaining that all the fibres of each optic tract pass across to the eye of the opposite side. The weight of authority and the best of the argument are still on the side of the older theory, and Knapp (Archives of Ophthalmology and Otology, vol. i. No. 2, p. 242,) in speaking of the experiments of Morgagni, Gudden, and Admük, says, "The results of

these experiments are: in the dog and cat, if one eye or optic nerve is destroyed, the atrophy strikes both optic tracts, but in the rabbit it strikes only the optic tract of the other side. The present phase of the question of the decussation is the following: In animals that have no common field of vision, the crossing is total; in others that have a common field, the crossing is partial, corresponding to the extent of the common field. In man, there is half crossing." As, however, the subject is still much discussed, it may be considered, to a certain extent, an open one; particularly when we find in so recent a publication as Mr. Carter's Ophthalmic Surgery the statement that "It was recently believed that the decussation was only partial, and that the external fibres of each tract went to the same side; but this view is not supported by the later investigations."

In fish, the arrangement is simple and plain enough; the right nerve crosses entire above the left, and, if it may be considered as demonstrated by physiological experiments that in animals of higher development, and with a common field of vision, there is a partial decussation, the manner in which this twisting of the fibres has been brought about furnishes a curious problem for evolutionists. As has been pointed out by Wollaston, however, though the anatomical analogy has not been maintained, the physiological analogy is still complete; for as, in the case of animals that have not a common field of vision, the axes of the eyes are directed outwards instead of forwards, the visual impression of objects on either side is conveyed to the opposite hemisphere of the brain, just as the

semi-decussation theory supposes it to be in animals with a common field.

There seem to be difficulties in the way of the microscopical study of the chiasm in man that prevent an agreement of authorities. Gudden insists that semi-decussation can be determined by a series of careful sections of the chiasm, while Mandlestamm and Michel claim that their anatomical researches demonstrate that the crossing is complete. This refers the question to clinical observation and pathological investigation, and may give an interest to the report of cases that bear upon it

The terms right and left hemiopia, as they are now universally used, are rather unfortunate, and may lead to confusion in the minds of students, while even those more familiar with the subject may often have to hesitate for a moment to translate them from their real to their accepted meaning. This confusion of terms becomes more marked when we remember that the terms hemeralopia and nyctalopia are used in a different sense. By hemeralopia we express the fact that the patient can see in daytime but cannot see at night, that he is night-blind; while by right hemiopia, on the contrary, we mean that the patient cannot see on the right side but can see on the left. Naturally, therefore, the expression "so-called" right or left hemiopia is sometimes met with. These terms can be made correct only by applying them to the retina instead of to the field of vision, for in right hemiopia the right side of the retina remains unaffected, and enables the patient to see to the left. By right hemiopia, therefore, we really mean right half-blindness.

The only entirely appropriate cases for the illustra-

tion of theories of decussation are those in which there is a sharp line of demarcation, symmetrical on the two sides, between the deficient and the unaffected portions of the visual field, and in which there is no suspicion that the symptom may be due to disease of the retina or disk, or of the optic nerve in front of the chiasm. For example, a patient recently presented himself at Wills Hospital with well-marked binocular hemiopia which at once suggested intracranial disease, but the ophthalmoscope showed an almost symmetrical retinal separation in each eye. This, of course, was a mere chance, but a chance not less likely to occur in the disk or in the anterior part of the nerve. Even in the chiasm, it seems hardly possible that any form of disease or of pressure from neighboring growths should accurately dissect out a certain set of fibres, leaving the others completely intact; so that a sharply defined, symmetrical, binocular hemiopia may be considered as furnishing a very strong presumption, at least, in favor of lesion of the optic tract or of the brain behind it.

The following two cases are fair illustrations of cerebral hemiopia.

J. H., sight good until four weeks before examination, when it became obscured suddenly. Had had diabetes three or four years ago, but sugar had disappeared entirely from urine, and health seemed restored. Had lately had some headache, feeling of weight on top of head, and slight mental confusion and loss of memory; the right side of the field of vision was deficient, almost obliterated to the median line in each eye. In the left side he could read ordinary print with a slowness and difficulty that seemed to be due partly to imperfect vision, and partly to loss of memory and of the power of mental concentration. There was no other paraly-

sis. The ophthalmoscopic appearances were normal, except a slight engorgement of the retinal veins and several small yellowish-white spots in the neighborhood of each macula, suggesting albuminuria; albumen was found in the urine in considerable quantities. Eight months afterwards, the edge of the obscure portion of the field had receded, and was three inches to the right of the point of fixation, at a distance of one foot from the eye. The line of demarcation was still sharp and vertical, and perfectly symmetrical in the two eyes, so that the same diagram accurately described the field in each.

The ophthalmoscopic appearances in this case were barely sufficient to excite a suspicion of the earlier stages of albuminuric retinitis, which, as yet, interfered but little with vision. The right hemiopia pointed to a lesion of the left hemisphere of the brain, while the albuminuria indicated a condition of the vessels that would predispose to hemorrhagic extravasation, and the mental symptoms confirmed the diagnosis of cerebral disease.

The second case has more interest, from the fact that it was complicated with other symptoms which located the disease almost positively on one side of the brain.

G. H., æt. 29, six months before his admission to Wills Hospital had chancre, followed by eruption on the face, sore throat, and other unmistakable, secondary, syphilitic symptoms. About four months after the appearance of the chancre he had violent headache, which continued in less degree; but, three days before admission, he read for several hours with ease, before going to bed. The next morning he awoke with dizziness and dimness of sight. Double vision was first noticed two days afterwards. The following condition was found on examination. On the right side

there were partial ptosis, complete paralysis of internal rectus, paresis of superior and inferior recti, and sluggishness of the pupil. There was complete left hemiopia on both sides, with fair vision in the right halves of both fields. No other symptom in the left eye but diminution of vision. O. D. V. $= \frac{20}{C}$? O. S. V. $= \frac{20}{L}$. The ophthalmoscope showed only engorgement of the retinal veins, more marked in the right eye. On rough examination, there seemed to be a perfectly vertical and symmetrical line of demarcation through the macula; but when the fields were carefully taken, this line was found to incline a little from left to right for each eye, more decidedly for the left, and to be a little to the left of the macula on each side. After several weeks of treatment by mercurial inunction and large doses of iodide of potassium, the vision was much improved—O. D. and O. S. V. $= \frac{20}{XXX}$, but there was no change in the hemiopia. A gummy tumor or other lesion might easily involve both the optic tract and the third nerve in their passage over the crus cerebri, or the filaments of both at their origin, without implicating any other nerve; but it is difficult to conceive how disease situated elsewhere than in the right side of the brain could produce this combination of symptoms.

Medical literature furnishes numerous instances of hemiplegia with paralysis of the half of each retina on the side opposite to that of the limbs, and Dr. Hughlings Jackson considers it an almost invariable rule that when bilateral hemiopia is found in connection with hemiplegia, the patient cannot see to the paralyzed side.

It must be admitted, however, that, with a fair amount of ingenuity and a sufficiently strong bias, most of the phenomena of hemiopia can, on merely theoretical grounds, be explained by either theory of decussation. Thus, on the total decussation theory, right-sided hemiopia may be referred to pressure on

the left external angle of the commissure involving the outer half of the left optic nerve and, at the same time, the outer half of the left optic tract, and, consequently, the inner half of the right optic nerve-fibres.

Bitemporal hemiopia may be accounted for by pressure upon the anterior angle of the commissure, involving the inner halves of the optic nerves, and nasal hemiopia by pressure at the posterior angle involving the inner halves of the optic tracts, and, therefore, the outer halves of the optic nerve-fibres.

On the semi-decussation theory, right- or left-sided hemiopia may be rationally explained by supposing a lesion of the optic tract of the opposite side, and binocular, temporal hemiopia may be referred either to pressure in front of the commissure involving the inner halves of the optic nerves, or, behind the commissure, involving the inner halves of the optic tracts. Binocular, nasal hemiopia, cannot be explained by any single lesion, and this has been considered the weak point of the semi-decussation theory. The cases of this form of hemiopia are, however, so few, that, to say the least, they may be considered exceptional, and the existence of double lesions, nearly symmetrical, is almost within the range of possibility. Schoen, writing in 1874, says, "Nasal hemiopia is excessively rare, if ever met with. There is no record of an entirely incontestable case." (Annales d'Oculistique, t. lxxii., p. 182.) I have not been able to find a detailed account of any case quite free from complications.

Some cases have been reported in which the lower half of both fields was absent, but I know of nothing

to disprove Mauthner's assertion that no well-described case with a sharply defined horizontal line is on record. Such a case might be explained, with about equal plausibility, on either theory.

Though the theoretical discussions, which have been considerably extended on this subject, are often extremely ingenious and always interesting, any decision reached through them is, after all, merely an opinion, and no observations are positively conclusive that are not confirmed by post-mortem examination. I believe that wherever post-mortem evidence is entirely unequivocal, it will be found on the side of semi-decussation.

When we remember the special prominence of *nasal hemiopia* in the discussion of this question, there seems a remarkable absence of this positive kind of evidence connected with it. The literature of hemiopia furnishes only four cases of this form of the affection, in which post-mortem examinations have been made. Though, with perhaps one exception, none of these cases are free from local complications of disease of the retina, or of the nerve near the ball, or of both, which, on a rigid sifting, might fairly rule them out, a brief account of them may have, at least, some historical interest.

The only one in which the ophthalmoscopic appearances are reported as normal, is recorded by Müller (quoted by Knapp, in Brown-Séquard's Archives). The symptoms were complicated, and it was as much a case of temporal as of nasal hemiopia. There was first temporal, binocular hemiopia, then total blindness, and then nasal hemiopia with well-defined lines. Post-mortem examination showed a tumor in the sella turcica as large as a medium-sized apple.

A second case is given by Schüle (quoted by Plenk, in Archives of Ophthalmology and Otology, vol. v. No. 2, p. 201). "The disease began with amblyopia in the right eye, atrophy of the papilla, with blue discoloration and marked reduction of eccentric vision; later, atrophy commenced in the left papilla, with a similar course and changes in the arterial vessels. Autopsy: the ventricles much dilated; the infundibulum and both nerves distinctly atrophied and of a grayish color." The same author also quotes a case observed by Wegner and Schmidt. The perceptive and non-perceptive portions of the visual fields were not divided by a sharply defined line. The ophthalmoscope showed neuro-retinitis. On post-mortem examination, a tumor was found in the median line of the brain, involving the infundibulum, fornix, and septum pellucidum. The microscope showed decided atrophic changes both in the nerves and retinæ. The fourth case is reported by Knapp, in Brown-Séquard's Archives. Vision was very much diminished throughout the whole field, the lines between the totally and the partially obscured portions were not quite symmetrical in the two eyes, and there was optic neuritis in both. On post-mortem examination, the brain was found healthy, but the arteries at its base atheromatous. The nasal hemiopia was referred by Dr. Knapp to paralysis of the outer half of each retina, resulting from compression of the external fibres of both the nerve and optic tract, on both sides, by the anterior cerebral and posterior communicating arteries. It must be admitted that this ingenious arrangement has not seemed quite so clear to the opponents of semi-decussation.

Binocular, temporal hemiopia, in which the inner halves of the retinæ are involved, is also a very rare affection; and beside that of Müller, mentioned above, in which temporal hemiopia preceded the nasal, I can learn of only one post-mortem examination in such a case. It is reported by Sämisch (Plenk, loc. cit.). In both of these cases there were sarcomatous

growths involving the anterior angle of the commissure, and the hemiopia, therefore, admits of equally satisfactory explanation on either theory.

Autopsies in cases of homonymous *bilateral hemiopia* have been more numerous, and their results more definite. It is worthy of note that in all the reported cases the lesion has been behind the chiasm, and therefore more strictly cerebral; these cases are in the nature of positive evidence, and, to my mind, are quite conclusive in favor of the semi-decussation theory.

The first case of hemiopia on record is reported by Wollaston, who was himself the subject of it. After his death, an examination of the brain revealed a lesion of the right optic thalamus, which is described as converted into a tumor as large as a hen's egg, harder than the brain, and somewhat of a caseous substance. (Mackenzie on the Eye, p. 931.) The connection of this tumor with the hemiopia, however, is far from clear, as the symptom, which occurred twice with an interval of twenty years, lasted only fifteen or twenty minutes each time, and the right halves of the retinæ were involved on the first occasion and the left halves on the second.

It seems more likely that this was one of those singular cases of transitory hemiopia, not very uncommon, due to some temporary cause, and not dependent on discoverable organic change.

The following post-mortem records, which are all that I have been able to find, are of quite recent date.

Two are reported in the Archives of Ophthalmology and Otology, vol. v. No. 2, one by J. Hirschberg, of Berlin, and one by Dr. T. R. Pooley, of New York. In the first, there

was "defined, symmetrical, right-sided defect in the visual field of both eyes, with a nearly normal acuity of vision and a normal ophthalmoscopic picture, accompanied by aphasia and hemiplegia of the right side. The autopsy revealed a tumor as large as an apple in the left frontal lobe, implicating the left optic tract." In Dr. Pooley's case, there were right-sided hemiopia, sharply defined in the vertical meridian, partial hemiplegia of right side, and choked disk in the left eye, but no change of appearance in the right. On post-mortem examination, a gummy tumor was found in the left posterior lobe of the brain. The left thalamus opticus and the neighboring brain-substance were completely softened. A third case is also referred to in the same journal (Jackson, quoted by Plenk). There was bilateral, left hemiopia, and the autopsy showed pronounced softening of the right optic thalamus.

In a case of left hemiplegia, left hemi-anæsthesia, and left hemiopia, Dr. Hughlings Jackson diagnosed disease of the right optic thalamus, and post-mortem examination showed softening confined to the posterior and inner part of the right optic thalamus. (Medical Times and Gazette, Oct. 28, 1876.)

Dr. Gowers also reports a case in which there had been hemiopia for several months before death, and in which post-mortem examination showed softening of the posterior tubercle of the optic thalamus on the opposite side. (Ibid.)

We may add to these, as approaching very nearly to an autopsy in the accuracy with which the lesion may be located, the case of gunshot injury of the head reported by Drs. Keen and Thomson (Photographic Review of Medicine and Surgery, Feb. 1871). The wound of entrance was in the middle line, one and a quarter inches above the external occipital protuberance, and the wound of exit two inches to the left of the median line, and three inches above the wound

of entrance. The patient was unconscious for several months, and had right hemiplegia for about a year. More than eight years after the injury, the field of vision was "found to be divided for each eye by a line passing through its centre, in the vertical direction: total blindness existing to the right, and perfect vision to the left, of this line." These cases need little comment; they are typical, uncomplicated instances of cerebral hemiopia, and their evidence is strong in favor of semi-decussation.

There is another form of hemiopia which it may be of interest to mention in this connection, though not enough is known of it to give it any real scientific weight in the discussion. I refer to those strange cases of transient hemiopia which are met with most frequently in subjects of migraine, in whom they seem to be a kind of prodrome of the headache, though they occasionally occur without any other symptom. We not only know nothing of their etiology, but even the symptomatology, derived of necessity from the accounts of patients, is not definite or accurate. Usually, merely the fact that only the halves of objects are seen is reported, and we are without the means of knowing whether, in frequently recurring cases, the same side of the retina is always affected. In Wollaston's case, opposite sides were affected in the two attacks, but the interval between them was so long that they can scarcely be considered as phenomena of the same disease. He relates the case of one of his friends, who had been habitually subject to this symptom for sixteen or seventeen years whenever his stomach was deranged, in whom the blindness was invariably in the right side. A medical gentleman who recently told me of his own

case, assured me that the blindness was always on the same side, and that the line of demarcation was vertical and sharply defined. The symptom occurred quite frequently during his student-days, and was always the forerunner of a raging headache.

Weeker (Maladies des Yeux, p. 384) speaks of recurrences of transient, bitemporal hemiopia, with sharp lines of demarcation, unaccompanied by cerebral symptoms, and explains them by supposing an insufficient flow of blood, something like what is thought to occur in epilepsy, in certain regions of the brain supplied by symmetrical arteries. This is an attractive theory, and would be satisfactory if it could be shown that such symmetrical regions of the brain only were always involved. A lady who comes occasionally to my office, has been subject, for several years, to attacks of *inferior hemiopia* coming on suddenly every four or five months, preceded by no warning, and followed by no other symptom. She is in good health, and is not of an hysterical or nervous temperament. The attack lasts fifteen or twenty minutes, during which the field of vision is bounded by a sharp, perfectly horizontal line, and all below this is in the blackness of darkness. She has sometimes been seized on the street, and, being unable to see her feet or the ground for some distance in front of them, has been greatly embarrassed to find her way home. I believe that this case, which gives no aid or comfort to either theory of decussation, is unique, and that, in the present state of medical science, it does not admit of intelligent explanation.

Doubtless there are still many more things in the anatomy and physiology of the brain than are dreamt of in our philosophy, and it must be admitted that

cerebral pathology is not, as yet, a good field for complacent dogmatism; since so many cases are on record in which the most definite and unmistakable post-mortem revelations have been preceded by symptoms of the wrong kind, or on the wrong side, or in which the entire absence of symptoms has bid defiance to all theories: but perhaps few of these theories rest on a firmer basis of physiological experiment, clinical observation, and post-mortem evidence, than that of the semi-decussation of the nerve-fibres in the optic chiasm.

Supposing the theory of semi-decussation to be correct, an interesting point in the minute anatomy of the retina, that must depend upon the study of hemiopia for its elucidation, is the distribution of the nervous filaments at the dividing line between the two halves of the retina. The observations upon the exact position of the line of demarcation in hemiopia have been too few, and not sufficiently in accord with each other, to be much more than suggestive: as illustrated by the first case reported above, this line may even vary very considerably at different stages of the disease, while still continuing perfectly symmetrical in the two eyes. It is a significant fact, though, that whenever observers have not contented themselves with stating that half the field of vision has been obliterated, but have been careful to define the limits of the obscured portion with accuracy, it is found that this portion has been a little less than half. This, as far as it goes, supports the theory of Hirschberg, that there is a retinal zone, of about six degrees, which would be halved by a vertical line drawn through the point of fixation, supplied in common from both optic tracts.

THE OPHTHALMOSCOPE AS AN AID TO MEDICAL DIAGNOSIS.

By

MORRIS J. LEWIS, M.D.,

ASSISTANT PHYSICIAN TO THE CHILDREN'S HOSPITAL.

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At the Pennsylvania Hospital, during the last three years, several very interesting cases showing pathological changes in the eye-ground have occurred in the service of Dr. James H. Hutchinson, who kindly places the notes which were taken at the time, at my disposal. I have studied the cases with him, during the first year as resident physician to the hospital, and during the remainder of the time as his clinical clerk. The assistance of Dr. W. F. Norris has nearly always been obtained in making the ophthalmoscopic examinations, and the post-mortem examinations have been made by Dr. Morris Longstreth.

In the Philadelphia Medical Times for May 8, 1875, Dr. Hutchinson published a case of supposed Ménière's disease, the notes of which, briefly summed up, are as follows:—

Case I.—Catharine C., aged 42, an unmarried seamstress, first noticed pain at the top of her head in the autumn of 1873; this was thought, at the time, to be due to nasal catarrh. In the following June she began to lose her hearing, and, in the course of a few weeks, became almost absolutely deaf; the later symptoms were tinnitus aurium, unsteadiness of gait, pains in the limbs accompanied by

impairment of sensation, vertigo, and occasional vomiting. In October, 1874, she was admitted into the hospital, and then a tendency to fall forwards and to the right, and almost constant vertigo while erect, were noticed. Headache was a very marked symptom, and there was also nausea. No history of syphilis could be obtained, nor was this disease suspected. An ophthalmoscopic examination, made soon after the patient's admission, showed the optic disks to be prominent and dull-red in color, with fulness and tortuosity of the retinal veins, which plainly dipped over the edge of the disk, and marked striation of the retina, but no hemorrhagic spots. O. S. fundus + $\frac{1}{6}$, summit of disk + $\frac{1}{1\,6}$; O. D. fundus + $\frac{1}{4}\frac{1}{6}$, summit of disk + $\frac{1}{2}\frac{1}{6}$.

Upon this case Dr. Hutchinson remarks as follows: "If this pathological condition [alluding to Ménière's disease] is really present, it is not sufficient by itself to explain the choking of the disk, which I believe is caused by some interference with the flow of blood within the cranial cavity;" and he concludes by saying, "At all events there is in the case a lesion different from that which is generally supposed to exist in Ménière's disease, which I think could only have been discovered by the ophthalmoscope."

In March, 1875, the patient was discharged somewhat improved, but in April, 1876, again returned to the hospital, where she died on June 1, of the same year, under the care of Dr. J. Aitken Meigs, by whose permission I publish the remainder of the notes. The patient was deafer than on her previous admission, and this was particularly marked in the left ear; she also had headache, nausea, and vomiting. Her urine at first was normal, but subsequently showed a trace of albumen; no casts, however, could be discovered. Later symptoms were great dizziness, so that she fell upon the least attempt to walk, and an increase of the old pain in the back and limbs. Shortly before the fatal event took place, she lost completely the power of motion in her legs, while sensation was not entirely destroyed. At no time was there

any facial paralysis, nor did she complain of impairment of vision, although she wore powerful convex glasses.

The autopsy revealed two sarcomatous masses, one situated over the internal auditory meatus of each temporal bone, and that on the left entirely closing the jugular foramen on that side; the foramen on the opposite side was twice its normal size. There was marked ventricular dropsy, with consequent bulging of the infundibulum. The chiasm was softened, apparently as a consequence of pressure. Upon removing the posterior segments of the eyeballs, together with the optic nerves, the nerve-sheaths were seen to be considerably distended just behind the sclerotic, and the intra-cranial portion of the left optic nerve was flattened and somewhat softened. The left optic papilla was prominent, particularly upon the nasal side, and two arteries were seen curving over its edge; while in the right eye the papilla was prominent only upon the nasal side, and the vessels were much more obscured, some being irregularly enlarged.

This case shows particularly the value of an ophthalmoscopic examination, for, until this had been made, the original diagnosis had satisfied all those who saw the patient; and, in addition, it refutes the statement, made by some physicians, that a diagnosis of intra-cranial disease can never be made with the aid of the ophthalmoscope, unless it can be equally made without the employment of that instrument.

I report the next case on account of the conditions discovered upon the post-mortem table, no examination of the eyes having been made during life.

Case II.—Charles S., colored, æt. 20, single, waiter. His family history was good; he was intemperate, but denied all venereal taint. Since the year 1872, the patient had had occasional pain in the shoulders, and in the following year he began to cough and to expectorate muco-purulent sputa.

In August, 1876, he was admitted to the hospital, suffering from slight, irregular chills, and intermittent pain on the right side of his head, limited to the area supplied by the trifacial nerve; during these attacks he complained of dimness of vision. He was naturally thought to be suffering from malaria, and was treated with large doses of quinine, under the use of which he rapidly improved, and was discharged in two weeks, cured of his neuralgia, but with dulness at the apex of the right lung, and slight cough.

In the latter part of October the patient exposed himself to cold, and was immediately taken sick; nausea, vomiting, constipation, œdema of legs, and distension of the abdomen appeared, and the cough became much worse. On his second admission, for he returned to the hospital, there was marked ascites, with pain in the hepatic region; and examination of the lungs showed dulness over the whole of the right lung, and slight dulness at the left apex, with subcrepitant rales generally diffused. There were no cerebral symptoms, and there was no return of the neuralgia. The diagnosis of tubercular peritonitis was made. The patient died Nov. 14, 1876, and, at the autopsy, in addition to marked tuberculosis of the abdominal and thoracic viscera, three tubercular masses, each about as large as a cherry, were found in the brain; one in the posterior part of the right hemisphere, near the cortex, a second in the left hemisphere over the posterior horn of the ventricle, and a third in the right lobe of the cerebellum towards the inferior surface. There was no meningitis, but the ventricles contained considerable fluid. Upon removing the posterior halves of the eyeballs, marked choking of the optic disks was discovered, and the optic nerve-sheaths were distended slightly, near the sclerotic. (This last observation was made after the eyes had been for some time in a solution of chromic acid.)

Had an ophthalmoscopic examination been made in this case on the patient's first admission, it is most probable that choked disk would have been recognized,

and attention would then have been called to the condition of the brain; as it was, this was overlooked, for the neuralgia was the only symptom that pointed to intra-cranial disease.

Case III.—In this connection I will refer to the case of Sarah M., reported by Dr. Hutchinson to the College, a few months ago; it is recorded as a "Case of malignant disease of the retro-peritoneal and mesenteric glands, rib, and vertebræ, etc." (vide *supra*, page 55), but there was also a cancerous mass the size of half a hen's egg, projecting into the cranial cavity from the left middle fossa, and making a deep depression in the anterior part of the left middle lobe; there were no cerebral symptoms whatever, and the ophthalmoscope showed normal optic disks. At the autopsy, no fluid was found in the ventricles, and the optic nerve sheaths were not distended. The case is particularly interesting in this respect as it tends to prove that choked disk may be caused by effusion into the intracranial cavities.

Case IV.—In a case of abscess of the brain, reported by Dr. Hutchinson to the Pathological Society last winter, ophthalmoscopic examination revealed slight choking of the right papilla, with white patches—the evidences of old hemorrhages—on the nasal side of the disk. On account of symblepharon the left eye could not be examined. The autopsy revealed an old abscess situated in the right optic thalamus, and marked dropsy of the ventricles. Upon removing the posterior parts of the eyeballs, together with their nerves, the papillæ were found choked, particularly the left; the optic nerve-trunks on both sides appeared normal, and "especially was it noted that no distension or abnormal looseness of the external sheath existed at or close to the selerotic."

In the following cases no autopsies were made, the patients either recovering and leaving the hospital, or, if they died, their friends refusing an examination.

Case V.—John B., æt. 21; single; porter; was admitted April 17, 1877. No hereditary taint was discoverable; the patient was moderately intemperate, but denied having had syphilis. During the summer of 1876, he had had a severe attack of "bronchitis," which had been accompanied by occasional, blood-stained expectoration, but he had recovered towards winter. Three weeks before his admission he had noticed anorexia, conjunctivitis, and photophobia, and, two weeks later, headache, pain in the extremities, and epistaxis appeared. On admission there was violent headache, marked irregularity of the heart's action, and constipation; but no lung complications were discoverable, and the urine was normal. Some of the symptoms pointed to typhoid fever, and in fact for a short time the patient was thought to be suffering from that disease, but, upon making an ophthalmoscopic examination, slight prominence of the papillæ (the outlines of which were indistinct), and marked fulness of the veins, were discovered. Attention was then more particularly directed to the cerebral symptoms, and, in the course of two days, pain in the back, shooting into the extremities, and slight retraction of the head, occurred, and it then became evident that the case was one of meningitis, probably cerebro-spinal. The further progress of the case verified this diagnosis. Hiccough, opisthotonos, delirium, marked hyperæsthesia, subsultus, retention of urine, and strabismus appeared, about in the order mentioned. Subsequent examination of the lungs merely revealed a little harshness of the respiration at the left apex. Vomiting only occurred twice. The patient died comatose on the thirteenth day after admission, but shortly before death an examination of the eyes by Dr. Norris revealed "choking of the left disk with haziness of the retina, and œdema of the right disk." No autopsy was allowed.

In the following case the ophthalmoscope only served to verify the diagnosis previously made.

Case VI.—Peter R., æt. 35; married; waterman; Irish; was admitted Jan. 10, 1874. Six months previously he had been sunstruck, but from this he had apparently recovered, and he considered himself in good health until a few weeks before applying to the hospital, when he began to suffer from headache, malaise, and vomiting. About January 1, he became delirious, and, soon after this, incontinence of feces made its appearance. On admission, the patient was almost comatose; there was marked rigidity of all his muscles, and forced movement apparently caused pain; there was also by this time incontinence of urine, but upon examination the latter was found to be normal. There was no cough. A short time after this the patient became rational, and was able to speak, but did not complain of headache. The diagnosis of "meningitis following sunstroke" was made. Examination of the eye-grounds revealed hazy, swollen disks, and distended and tortuous veins. The patient was given iodide of potassium, but gradually sank, and died February 17, 1877, the "Cheyne-Stokes" character of respiration having become marked towards the close of life. In this case also no autopsy was allowed.

Case VII.—In the Philadelphia Medical Times for May, 1875, Dr. Hutchinson reported a case of cerebro-spinal meningitis, in which the cerebral symptoms were so slight that it was doubtful whether the meninges of the brain were implicated or not, or in fact whether the patient was not merely hysterical, as at one time she jumped out of bed to see a passing parade; but upon examination with the ophthalmoscope, marked choking of the disks, together with retinal hemorrhages, was discovered, which made the diagnosis of cerebro-spinal meningitis almost a certainty when the other symptoms were taken also into consideration. The further progress of the case showed the correctness of the diagnosis. The patient recovered perfectly, all traces of the choking eventually disappearing.

It is not in well-marked cases of meningitis, where there is no doubt about the diagnosis, that the oph-

thalmoscope is most valuable, but in the obscure forms, particularly in the tubercular meningitis of children, and in meningitis complicating the exanthemata, where the outset is so often insidious. In metastatic meningitis, Huguenin considers choked disk as one of the most reliable of all signs. Ziemssen states that the stiffness of the neck may be entirely wanting in cases of cerebro-spinal meningitis, and Paonlucci even reports a case, verified by autopsy, in which there were no cerebral symptoms whatever; but neither of these authors reports the condition of the eye-ground.

The majority of intra-cranial growths are syphilitic, and energetic treatment, if commenced in time, will in the majority of cases effect a cure; so that but few of these find their way to the post-mortem table. The two following cases were undoubtedly of this nature:—

Case VIII.—Henry C., æt. 41; single; nurse; Irish; was admitted January 2, 1875. When 20 years of age he contracted chancres, and again eight years later. In 1868 he was treated in the Pennsylvania Hospital for venereal warts, and since then has served as an assistant nurse in the hospital. He has had frequent attacks of facial erysipelas. Five weeks before admission into the Medical Ward, he noticed vertigo and unsteadiness of gait, and a few weeks later the vision of his right eye began to fail; after this slight paralysis of the right side of the face appeared, accompanied by marked sluggishness of mind, but not by headache. The patient then had a violent convulsion, which was followed by unconsciousness for about ten minutes; upon recovering, he had intense occipital headache, ptosis of the left lid, increased paralysis of the right side of the face, tinnitus aurium, and deafness. There was no hemiplegia nor

aphasia, and his urine was non-albuminous. Ophthalmoscopic examination showed choking of the optic disks. Under large doses of iodide of potassium he almost entirely recovered, and in about one month the choking of the disks had given place to slight atrophy.

Case IX.—Simon M., æt. 35; single; sailor; German; was admitted September 10, 1875. About the year 1868, the patient had several attacks of unconsciousness, which from his description resembled *petit-mal;* but from these he recovered, and shortly afterwards contracted gonorrhœa, and probably a chancre, which was followed by phagedenic ulceration. He was treated very energetically for this in Shanghae, and was severely ptyalized; his left arm then became exceedingly hyperæsthetic, and finally rigid and slightly atrophied. In the winter of 1873, violent pain of the left side of the head appeared, together with several nodes upon the temporal and parietal bones; dimness of vision and vertigo were also present. From this time until his admission, the patient had been exceedingly intemperate, and on that account was unable to give further information as to his previous history, having been almost constantly intoxicated.

On admission, there was found paralysis of the left side of the face, total anæsthesia of the conjunctiva on that side, tinnitus in the left ear, loss of sense of taste on the left side of the tongue, and impairment of the sense of smell. The left arm and leg were very slightly paralyzed. There was diplopia, and ophthalmoscopic examination revealed choking and haziness of the inner half of each disk, with commencing atrophy of the outer halves. Under the use of large doses of iodide of potassium, he improved greatly, and was discharged in April, 1876.

In the following case, the diagnosis was never positively made out, but the probability is that there was syphilitic disease of the spinal cord, with extension of inflammation to the brain.

Case X.—John R., æt. 36; widower; bricklayer; Pennsylvanian; was admitted February 28, 1877. His mother had died of apoplexy, but the rest of his family history was good. He had been a very moderate drinker. During the war he had served as a sharpshooter, and his sight was uncommonly good. Four years before admission he had contracted a chancre, but this he said had not been followed by bubo nor by any evidence of constitutional infection. In December, 1876, he began to suffer from headache, fever, and pain in the left side upon full inspiration; but with this he had no cough. The headache lasted four weeks, was always worse at nights, and was at times excruciating. After this he had violent shooting pain in the left thigh and hip, and then his knees, left elbow, and shoulder became stiff and painful.

On admission, the pains persisted, and a very sensitive spot was discovered over the dorsal vertebræ; slight atrophy of the left deltoid muscle was also detected. There was a slight apex-systolic murmur. The urine was normal. At first the patient was thought to be suffering from chronic rheumatism, and was treated accordingly, but one month after admission, no improvement having occurred, an ophthalmoscopic examination was made with the following result: In the right eye, the disk was swollen upon the nasal side, and its outlines were indistinct; the veins were very full and tortuous. In the left eye, the disk was more swollen, being seen with $+ \frac{1}{3}\frac{1}{3}$, and the surrounding retina was hazy; the veins were still fuller than in the other eye. He was given large doses of iodide of potassium, and in the course of ten days was able to walk quite well, whereas before he could scarcely leave his bed. In May, just before he was discharged, a re-examination of his eyes revealed greater indistinctness of the disks, and greater fulness of the veins; there was also noticed for the first time slight internal strabismus of the left eye. During the patient's stay in hospital, he had no headache, nor other symptoms pointing to intra-cranial disease, except the condition of the eye-ground and the sub-

sequent strabismus; so that in this case the ophthalmoscope was of considerable service.

In the following case it is probable that the diagnosis of simple epilepsy would have been made, had not the ophthalmoscope been employed, and indeed this diagnosis had been made by the physician who sent the patient to the hospital.

Case XI.—William H., colored, æt. 26; laborer; was admitted December 14, 1874. He considered himself to have been perfectly well until the year 1873, except for an attack of gonorrhœa the year before; he denied, however, ever having had a chancre. In the spring of 1873 he had had an epileptiform seizure, and this had been repeated once or twice every week until the date of his admission to the hospital. He had at no time had an aura, headache, or vertigo. On admission there were found no paralytic symptoms, and no general or local anasarca. Ophthalmoscopic examination revealed marked choking of the disks, which were deep red in color; the veins were swollen and tortuous, and there were several hemorrhagic spots upon the retinæ, which latter were quite striated; shortly afterwards slight insufficiency of the right internal rectus was detected. Examination of the urine showed a slight amount of albumen, but at first no casts could be detected, though later a few granular and hyaline casts were found. During the month in which he remained in the hospital, the patient had three severe convulsions, which came on suddenly without the least warning, lasted about ten minutes, and apparently affected every muscle in the body; almost immediately after the seizures he would feel as well as usual. This patient unfortunately was lost sight of; it is probable that there was some intra-cranial lesion, possibly syphilitic, in addition to the Bright's disease, although it is possible that the latter condition gave rise to the choking, as it sometimes, although very rarely, does so. The lesion of the eye-ground in ordinary epilepsy, according

to Albridge (West Riding Asylum Reports, vol. i.), is merely hyperæmia of the optic disks and retinæ, and in some few cases active congestion; but in these cases this abnormal condition subsides somewhat in the intervals between the paroxysms. In syphilitic epilepsy, on the other hand, according to Fournier, there is nearly always "optic neuritis." The presence of the latter condition in the foregoing case is therefore in favor of the diagnosis of syphilis, while, on the contrary, the statement of Charcot that syphilitic epilepsy is most always hemiplegic in nature, is against it. The diagnosis therefore must remain somewhat doubtful.

A second case of albuminuria, in which the ophthalmoscope revealed changes closely resembling those found in cases of intra-cranial disease, also occurred in the hospital, and is worthy of record.

Case XII.—Jacob E., æt. 35 (?); married; steward; was admitted March 3, 1877. He had been a moderate drinker, but denied all venereal taint. In 1870 he had had a violent attack of renal colic, and a second one in 1874; in the interval he had felt moderately well, but had noticed that he was obliged to micturate very frequently. Soon after the second attack of colic he had observed slight œdema of his face and legs, and this symptom continued until his death. He never noticed any sandy deposit or calculus in his urine. On admission, the patient's legs were slightly œdematous, and he complained of violent headache, but had no impairment of vision. His urine contained a moderate amount of albumen and pale granular casts; the amount passed in twenty-four hours measured six pints. After remaining in the hospital for over two weeks, without any untoward symptom, the patient went out on a pass, and returned suffering from marked dyspnœa and cough; examination of the lungs revealed numerous moist, bronchitic râles. Ophthalmoscopic examination showed, in the right eye, the "disk bluish-gray, and wanting in capillarity, the upper part much swollen, looking as if it had been choked, and its outlines obscured; the fibre-layer

of retina opaque, and the retinal vessels diminished in calibre; and in the left eye the disk whitish, more swollen than that in the right eye, its outlines obscure, and the fibre-layer of the retina hazy; no retinal hemorrhages nor choroidal changes in either eye." The patient's condition became rapidly worse, and he died in convulsions three days after the exposure. The autopsy revealed marked granular degeneration of the kidneys and hypertrophy of the heart. The head unfortunately was not examined.

Several other interesting cases have occurred in the hospital: these, however, I will not report, for fear of wearying my hearers, but will proceed at once to make a few remarks as to the manner of causation of choked disk, and as to its value as a diagnostic sign.

The questions, What precise value is to be placed upon the ophthalmoscopic appearances in the diagnosis of intra-cranial diseases? and, What are the causes of the pathological conditions so discovered? have not yet been definitely answered, although there has been no want of diligent research in this direction by competent observers. Those who first used the ophthalmoscope, in cases of cerebral disease, were much more positive in their statements as to the differential diagnosis between the pathological changes discovered in the eye-ground, and their relative value as diagnostic signs, than subsequent observers; thus, Dr. Clifford Allbutt defines choked disk or ischæmia papillæ as the signal of intra-cranial tension, and neuritis as the signal of intra-cranial irritation, and states that these can be differentiated. Dr. Broadbent inclines in the same direction, but is held back by the doubts of Dr. Hughlings Jackson and Mr. Jonathan Hutchinson. MM. Raymond and Bouchut believe too that these lesions are essentially different.

Acknowledging for the sake of argument the difference between the conditions, a second difficulty arises, for inflammatory and obstructive changes in the disk are frequently associated, either being the consequence of the other; both conditions therefore being present at the same time, it would manifestly be impossible by mere ophthalmoscopic examination to state which was the primary trouble, though this may be said not to be an objection to the theory, but rather a proof of the immature state of our knowledge of this subject. Mr. R. Brudenell Carter, in his work on Diseases of the Eye, remarks that he has never been able to satisfy himself of the value of any ophthalmoscopic sign as showing the presence of inflammation, or the preponderance of inflammatory over obstructive changes. The consequence is that the majority of the later observers do not pretend to recognize which condition is the primary one, or even to recognize one from the other, but speak of optic neuritis and choked disk as synonymous terms. This is quite unfortunate, and, quoting Dr. Broadbent, "till this question is decided we cannot so much as pass the threshold of the inquiry as to the mode of production of optic changes in diseases of the brain." It is, however, positively determined that the ophthalmoscope is a very valuable aid in the study of cerebral disease, and is frequently the means of making a correct diagnosis.

According to Allbutt, "it is sufficient at present to say that the occurrence of papillary ischæmia or of optic neuritis, in any person, ought to awaken the gravest suspicion of cerebral disorder, and that the occurrence of simple atrophy should suggest great

watchfulness; the precise degree of meaning to be attached by the physician to optic neuritis cannot, however, be determined until a certain number of cases of optic neuritis without cerebral changes have been traced to the post-mortem table." Although this sentence was written in 1871, it still applies with full force, and will apply until the physician and the ophthalmologist work in concert. In the Lancet, for May 12, 1877, Dr. Jackson, in an interesting paper, calls attention to the fact that Mr. Couper has found that, in some cases of hypermetropia, the optic disks, in consequence of overuse of the eyes, become reddened and even swollen. The redness and swelling are sometimes so marked that this comparatively unimportant condition, remediable by spectacles, is occasionally mistaken for, and treated as, slight neuritis depending on cerebral disease.

In many cases of intra-cranial tumor, where amaurosis is a prominent symptom, atrophy of the disk is found upon examination, and thus has been looked upon by some as being the most common pathological condition of the eye-ground associated with brain tumor; if, however, an early examination is made, choked disk will frequently be found to be the precursor of this atrophic change. Jaccoud, in his "Pathologie Interne," 1877, alludes to 89 cases of brain tumor reported by Lebert and Friedreich; in these, there was amaurosis in 66, a percentage of 74.15. In the 91 cases in Anmuske's list, in which the condition of the sight was noticed, amaurosis occurred in 73, a percentage of 80.21. As will be seen later, this percentage is not as high as the percentage of changes in the eye-ground in intra-cranial disease;

choked disk may, therefore, be overlooked if the ophthalmoscope is only used in the cases attended with impairment of vision. According to Wadsworth, "atrophy of the disk in all these cases is to be regarded as the result of, and therefore as having the same significance as, neuritis." As regards the frequency with which choked disk and atrophy occur in connection with intra-cranial tumors, Annuske, in Graefe's Archives, and Reich, in a Russian medical journal, report 92 cases verified by autopsy, in which "optic neuritis, or *atrophia-ex-neuritide*," was found in 88 of the cases, being 95.65 per cent. In looking over the medical journals from the year 1874, the date of Reich's report, to the present time, I have been able only to collect 31 cases in which ophthalmoscopic examinations have been made, and in which the diagnosis has been verified after death; to these I add the 3 cases observed in the Pennsylvania Hospital, making 34 in all. Choking of the disk, or atrophy, occurred in 27 of these; in 3 there was redness of the disk, or dilatation of the retinal veins; and in 4 the disks were healthy; adding these to the cases previously mentioned, we have a total of 126, in 115 of which choking or atrophy occurred, a percentage of 91.26; but if the 3 cases in which "redness of the disk or dilatation of the vessels" was noted, be added and considered as cases of commencing choking of the disk, the percentage rises to 93.65. I annex the list to this paper. Annuske believes that these changes will be almost always, if not always, found, and thinks that the statements that intra-cranial tumors often do not give rise to optic neuritis, are the result of imperfect or insufficient observations.

It is now, however, positively determined that these exceptions do occasionally occur. (See Case III.)

It is not only in cases of brain tumor that the ophthalmoscope reveals morbid changes, but also in any condition that produces increased intra-cranial tension; this is particularly the case in the various forms of meningitis with ventricular dropsy; and, indeed, according to the statistics at present obtainable, the percentage of changes in the eye-ground in this condition is higher than in brain tumor. Thus in the 90 cases of meningitis reported by Bouchut and Heinzel, neuritis, or atrophy, was found in 94.33 per cent. of the cases. Dr. Hock (Archives of Ophthalmology and Otology, vol. v. No. 1) differs from the majority of observers as to the appearances of the fundus of the eye in meningitis, and states that, in his experience with cases of basilar meningitis in children, he has found the retina to be the part principally affected, and the optic disk rarely if ever at all swollen; he, however, draws no conclusions. Bartalot (London Medical Record, Aug. 15, 1876) arrives at very much the same opinion after observing carefully 24 cases. According to the former observer, infants show a greater predisposition than adults to the alterations of the optic nerve in the course of cerebral disease.

The second question, viz., What are the causes of choked disk? is not as easily answered as the first. According to Wadsworth (British Medical Journal, Nov. 5, 1874), "the direct progression of inflammation from the base of the brain along the optic nerve to the eye, has been anatomically proven only in rare instances;" and Pagenstecher (London Ophthalmic

Hospital Reports, vol. vii.) states that he has never been able to satisfy himself that the morbid process has originated in this manner, and that the more he has employed himself in researches of this kind, the more doubtful has he become as to this mode of origination of the changes met with in the disk; he strengthens his statement by reporting carefully seven cases, in four of which the "structure of the optic nerve, so far as it was examined, was perfectly normal," and in three other cases, "along with complete atrophy of the disk, there was also atrophy of the optic nerves all through the portions which had been removed for examination." These changes in the nerve, he therefore looks upon as being a consequence and not a cause of the choking.

The mechanical theory finds the most supporters at the present day, although the precise method of the origination of the obstruction to the return of blood from the eye, is still a matter of dispute. Von Graefe explains it by an increased pressure within the cranium retarding the return of blood by pressure upon the cavernous sinus, direct or indirect, which thus causes a venous stasis within the eyeball. Michel believes that the pressure on the sinus is brought about by ventricular effusion distending the infundibulum, and, as a consequence, interfering with the circulation in the cavernous sinus. An objection to these views arises in the fact that the sinus has quite rigid walls, and that the amount of pressure necessary to compress it would have to be far greater than that which is ordinarily found in intra-cranial disease; and, besides this, Seseman has shown that a free communication exists between the facial and the ophthal-

mic veins, which is sufficient to remove all stasis of the retinal veins, even should the sinus be occluded.

Schwalbe, by experiments, has proved that it is easy to distend the permeable space existing between the two sheaths of the optic nerve, from the arachnoid cavity, and that the fluid collects around the insertion of the optic nerves; further experiments by Manz and Schmidt show that the fluid finds its way into both optic nerves, although only one arachnoid cavity be injected. Axel-Key states further that the ventricles of the brain are in connection with the subarachnoid space, and that this latter space connects with a second space between the inner sheath of the nerve and the nerve itself.

In Dr. W. F. Norris's interesting paper on Optic Neuritis, read before the American Ophthalmological Society at Newport, July, 1874, after some allusions to the inflammatory changes being almost limited to the region of the lamina cribrosa, is the statement: "we have found, however, that although most intense at that point and in its vicinity they were not limited to it, but extended backward throughout the orbital portion of the optic nerve. This we can readily account for, when we consider that Schwalbe, as well as Axel-Key and Retzius, have demonstrated that fluids injected into the subvaginal space readily pass into the lymph spaces in the interior of the nerve, which surround and separate the individual nerve-fibres, thus producing throughout its length œdema and nutritive hyperplasia." From these statements it is seen that an excess of fluid in any of the cavities of the brain will distend the sheaths of the optic nerve, and thus compress the retinal vein at its emergence, and before

it joins the ophthalmic vein, besides interfering with the nutrition of the optic nerve itself, which, as is well known, is supplied directly from the cerebral circulation, and not from the vessels of the eyeball.

Hitzig states that during life, under normal circumstances, the subdural space contains cerebro-spinal fluid, so that it is even possible for intra-cranial tension to distend the optic nerve-sheaths by the displacement of this fluid, even though no abnormal amount is present. In four out of Pagenstecher's seven cases, this distension was very marked, and in two others it was present, though to a much less degree; but he considers that this effusion is due to local causes. Manz has discovered this so often that he considers that it must be an almost invariable occurrence in intra-cranial disease. Dr. Norris, in the paper before mentioned, reports two cases in which there was marked dilatation of the nerve-sheaths, coinciding with ventricular dropsy. In Cases I. and II., recorded in this paper, this condition was present; in Case III. it was absent, but here there was no ventricular effusion, and no choking of the disk; in Case IV., although there was choking, the notes say that there was no sheath dilatation. Abadie (L'Union Médical, Sept. 24, 1874) says, on the other hand, that he has rarely found this condition present.

It is stated that choked disk is most frequently found in association with growths occupying the cerebellum and the posterior parts of the brain; the probable explanation of this fact is to be found in the greater likelihood of interference with the flow of blood in the sinuses and venæ Galeni, by growths in this position, and, as a necessary consequence, of effu-

sion into the cavities of the brain. Benedict considers that an irritation conveyed by the sympathetic to the optic disk, causing an increased vascularity of the parts, is the main factor in producing choked disk, and alludes to his experiments in this direction with the constant current. In this, Pagenstecher follows him. Abadie calls attention to the experiments made by Claude-Bernard, in which section of the trifacial, behind the Casserian ganglion, caused ulceration of the cornea, conjunctiva, and tongue, although upon examination of the nerve trunks they were found to be perfectly normal; and suggests that the changes in the optic disk may be explained in an analogous manner. These last two theories however have very few followers.

In some cases of Bright's disease, a condition in no wise distinguishable from choked disk is found, as in Cases XI. and XII. I do not refer to the well-known retinitis albuminurica. In only one such case, however, can I find the record of a post-mortem examination, and in that case the brain is said to have been normal. It is reported by Mr. R. Brudenell Carter. Much valuable information can be obtained by examining the eye-ground in cases of Bright's disease. The discovery of pronounced retinitis, or of marked retinal hemorrhages, should lead one to make a grave prognosis as regards length of life. Mr. Bader (Guy's Hospital Reports, 3d series, vol. xii.) remarks, upon the subject of retinal hemorrhage, "we look upon the latter as a general warning as regards the state of the patient's bloodvessels;" and Mr. Carter has found that in his experience the termination of such

cases, among hospital patients, has been comparatively rapidly fatal.

In this connection the subject of cerebral hemorrhage naturally arises, and as the diseased condition of the arteries, which is generally the cause of the hemorrhage, is frequently associated with renal disease, the condition of the retinal arteries and of the retina may be of great diagnostic value. In looking over the Pennsylvania Hospital case-books for the past three years, I find that every case of cerebral hemorrhage was associated with contracted kidneys and hypertrophied heart. Unfortunately, however, no ophthalmoscopic examinations were made before death, nor were the eyes examined after that event took place.

While believing that the ophthalmoscopic signs are of great value in forming a correct diagnosis of cerebral disease, I do not think that they have the transcending significance which Bouchut, in his recent publications, attributes to them; but I believe that they should always be considered in connection with the other symptoms, and that the physician who does not use the ophthalmoscope in these cases is neglectful of one of the most important aids in the elucidation of a difficult subject.

TABLE OF THIRTY-FOUR CASES OF INTRA-CRANIAL TUMOR, IN WHICH OPHTHALMOSCOPIC EXAMINATION WAS RESORTED TO AS A MEANS OF DIAGNOSIS.

	Observer.	Character and position.	Condition of ventricles, etc.	Condition of vision.	Condition of optic nerves, etc.	Remarks.
1	Hughlings Jackson, Med Times and Gaz., Jan. 3, 1874.	Tuberculous mass in pons Varolii.	Ventricles and arachnoid space distended with fluid.	"Diplopia."	"Redness of disk."	
2	Id.: Ibid., Feb. 7, 1874.	Medullary glioma in pons Varolii.	Ventricles distended with fluid.	Not mentioned.	Double optic neuritis.	
3	Id.: Ibid., Feb. 7, 1874.	Sarcoma; surface of right hemisphere.	Not mentioned.	Not mentioned.	Double optic neuritis.	
4	Id.: Ibid., Feb. 28, 1874.	Right hemisphere.	Not mentioned.	Not mentioned.	Optic neuritis, left eye.	Optic nerve-sheaths distended.
5	Id.: Ibid., Aug. 1, 1874.	Gumma; middle lobe of cerebellum.	Ventricles distended with fluid.	"Failure of sight."	Double optic neuritis.	
6	Id.: Ibid., Oct. 24, 1874.	Ant. convolution and both lobes of cerebellum.	Ventricles distended with fluid.	"Sight good."	Atrophy.	
7	Id.: Ibid., May 1, 1875.	Glioma; middle lobe of cerebellum.	Not mentioned.	Blindness.	Double optic neuritis.	
8	Id.: Brit. Med. Journ., March 24, 1877.	Middle lobe of cerebellum.	Not mentioned.	Not mentioned.	Double optic neuritis.	
9	Broadbent, Ibid., Feb. 28, 1874.	Gumma; Fissure of Sylvius.	Not mentioned.	Impairment of vision.	Double optic neuritis.	
10	J. Shingleton Smith, Ibid., June 6, 1874.	Glioma; right lobe of cerebellum.	Not mentioned.	Sight good.	Double optic neuritis.	
11	Ramskill, Med. Times and Gaz., May 23, 1874.	Glioma; left hemisphere, corpus striatum, and optic thalamus.	Not mentioned.	Impairment of vision.	Commencing optic neuritis.	
12	Russell, Ibid., May 16, 1874.	Cancer; ant. lobe of right hemisphere.	Not mentioned.	"No diplopia."	"Optic disks healthy; veins too full."	
13	Headland, Ibid. May 30, 1874.	Sarcoma of both hemispheres.	Not mentioned.	Impairment of vision.	Double optic neuritis.	
14	Buzzard, Ibid., July 11, 1874.	Tubercle of cerebellum and medulla oblongata.	Not mentioned.	Sight good.	Double optic neuritis.	
15	Raymond, Gazette Med., Juin, 1874.	Cerebellum.	Not mentioned.	Impairment of vision.	Atrophy.	
16	Bull, Philad'a Med. Times, May 15, 1875.	Middle lobe of cerebellum.	Not mentioned.	Not mentioned.	Optic disks normal.	
17	Dreschfield. Brit Med. Jour., April 3, 1875.	Cancer; at base.	Not mentioned.	Sight completely lost.	Optic disks normal.	
18	Russell, Ibid., March 27, 1875.	Gumma; at vertex, median line.	Not mentioned.	Not mentioned.	Double optic neuritis.	
19	Duffin, Ibid., June 24, 1876.	Glioma of corpora quadrigemina.	Ventricles distended with fluid.	Sight lost.	Double optic neuritis.	Venæ Galeni compressed.
20	Dowse. Path. Society, Lond., 1876.	Gumma; torcular Herophili.	Not mentioned.	Sight lost.	Double optic neuritis.	
21	Id.: Ibid., 1876.	Glio-sarcoma; ant. wall of corpus callosum.	Not mentioned.	Sight good.	"Disks hazy."	
22	Gowers, Ibid., 1876.	Gliomatous tumor of left cerebral hemisphere.	Not mentioned.	Not mentioned.	Double optic neuritis.	

	Observer.	Character and position.	Condition of ventricles, etc.	Condition of vision.	Condition of optic nerves, etc.	Remarks.
23	Hulke, Med. Times and Gaz., Jan. 20, 1877.	Sarcoma; ant. and middle fossa.	Not mentioned.	"Confusion of sight."	Double optic neuritis.	Cavernous sinus destroyed.
24	Fox, Lancet, Jan. 6, 1877.	Glioma; left lobe of cerebellum.	Ventricles distended with fluid.	Impairment of vision.	O. D. optic neuritis. O. S. atrophy.	
25	Pooley, Arch. Ophthal. and Otol., vol. v., No. 2.	Gumma; left post. lobe of cerebellum.	Right ventricle greatly distended.	Impairment of vision.	O. S. choked disk.	Chiasma and nerve trunks normal.
26	Habershon, Guy's Hosp. Reports, 3d S. vol. xx., 1875.	Inflammatory growth in right post. lobe of cerebrum.	Left ventricles dilated.	Impairment of vision.	Congested disk with hemorrhagic spots.	
27	Id.; Ibid., 1875.	Tubercular masses in left side of pons and cerebellum.	Not mentioned.	Impairment of vision.	Optic neuritis.	
28	Id.; Ibid., 1875.	Sero-cyst of cerebellum, median line.	Ventricles much dilated.	Total blindness.	Choked disk.	
29	Norris, Amer. Ophth. Society, July, 1874.	Sarcoma of cerebellum.	Ventricles much dilated.	Impairment of vision.	Choked disk.	Optic nerve-sheaths distended.
30	Dentan, Arch. Gen. de Med., Dec. 1876.	Glio-sarcoma; front left frontal convolution.	Ventricles much dilated.	Not mentioned.	"Retinal vessels somewhat dilated."	
31	Kohts, Virchow's Archiv, Bd. lxvii.	Post. part of corpora quadrigemina.	Ventricles much dilated.	Not mentioned.	Double optic neuritis.	
32	Hutchinson, Present Paper, No. I.	Fibro-sarcoma of both middle fossæ.	Ventricles much dilated.	Not mentioned.	Choked disks.	Optic nerve-sheaths distended; left jugular foramen closed.
33	Id.; Ibid., No. II.	Tubercle; right and left hemispheres and right lobe of cerebellum.	Small amount of serum in ventricles.	Occasional dimness of vision.	Choked disks (found post-mortem).	Optic nerve-sheaths distended.
34	Id.; Ibid., No. III.	Cancer; right middle fossa.	No distension of ventricles.	Not mentioned.	Disks normal.	

ON THE

TREATMENT OF OLD DISLOCATIONS OF THE SHOULDER BY SUBCUTANEOUS SECTION OF THE HUMERUS AND THE FORMATION OF A FALSE JOINT.

WITH THE REPORT OF A CASE IN WHICH THIS OPERATION WAS PERFORMED.

By

J. EWING MEARS, M.D.,

SURGEON TO ST. MARY'S HOSPITAL, ETC.

[Read July 4, 1877.]

In the month of August, 1875, J. R., æt. 38, a resident of Trenton, N. J., consulted me for the relief of great pain and immobility of the right shoulder-joint. The following history was given at the time: In the latter part of June, 1874, the patient fell through a trap-door, striking the right shoulder on the steps as his body descended. Immediately after the receipt of the injury, the parts were examined by a physician, who placed the arm in a sling, and ordered sorbefacient lotions to be applied. Under this treatment the contusion and swelling disappeared in a short time, leaving the joint painful and restricted in its movements; there was also observed an inability to approximate the arm closely to the side of the trunk. At the expiration of a month the arm was removed from the sling, and all treatment was discontinued; the pain and

impairment of motion in the joint continued to increase; the former was of a dull, aching character, most distinctly felt just beneath the coracoid process of the scapula, and extending about half way down the arm, and was very marked at the close of the day's work. At night the patient experienced relief by removing the arm as far as possible from his body—as nearly to a right angle as he could bring it. Becoming anxious with regard to the condition of his arm, and desiring relief from the pain he suffered, he came to the city in quest of further advice.

An examination of the joint was carefully made, the clothing having been removed, and all of the signs of a luxation were found to be present. The arm was markedly projected from the side of the body, and extended, the elbow drawn backward, and the forearm in a state midway between supination and pronation. The shoulder was prominent, and the depression beneath the acromion process was quite conspicuous. The patient was unable to place the hand on the opposite shoulder while the arm was approximated to the side of the body. The head of the humerus could be felt beneath the coracoid process of the scapula, fixed in its abnormal position. As imparted to the hand, the temperature of the arm was lower than that of the other arm; as compared with the pulsation of the vessels at the wrist of the left arm, the beat was notably lessened in fulness and force in the vessels of the disabled arm. In the forearm, the muscles appeared to be normal as to texture and firmness, whilst those of the arm were soft and flabby, and in the deltoid muscle there were evidences of atrophic changes. Slight numbness

was experienced in the inside of the arm, forearm, and little fingers, corresponding to the parts supplied by the ulnar nerve.

The clothing of the patient having been put on, the movements of the arm were observed. With great effort, and by drawing back the shoulder, the patient could introduce the hand into the pocket of the pantaloons; the hip pocket could not be reached; the watch could not be taken from the vest pocket on the left side; all of the manipulations necessary in putting on and removing articles of dress, were performed with the sound arm. The patient was unable to button his shirt-collar, or use a hair-brush with the affected arm.

With a view to extend the examination, and to ascertain the possibility of effecting reduction, I suggested the administration of ether on the day following. To this proposition the patient assented, and the anæsthetic agent was given by Dr. C. E. Black, U. S. Navy. Before he was placed under its influence, I distinctly stated to the patient that my object was to confirm the diagnosis which had been made, and to determine whether it was advisable to make any efforts at reduction, rather than to attempt by forcible manipulations to restore the head of the bone to its normal position. I put before him the danger of violent efforts at replacement in old luxations, and informed him that he must not be disappointed if I should make no extended efforts at reduction. When completely anæsthetized, a careful examination was made, and the diagnosis was confirmed. The head of the humerus was found to be fixed in its acquired position by adhesions, which

existed to such an extent as to make any efforts at reduction inadvisable. Very little more, if any, movement was attainable under the influence of the anæsthetic agent than was accomplished by the unaided efforts of the patient himself, thus showing that he had in his daily work developed the use of the crippled arm to its fullest extent. When he had recovered from the effects of the ether, I informed him that the parts had been found in such a condition as to render efforts at reduction hazardous, and that he must endeavor to content himself with the arm in its present, impaired state. He was advised to maintain as much motion in the arm as possible, and to apply anodyne lotions to control the pain.

In December of the same year, the patient again presented himself, and stated that the pain had increased, and that the movements of the arm had become more restricted. He earnestly desired that something should be done to afford him relief from the wearing pain, and at the same time, if possible, to restore his arm to its former usefulness. I refused to make efforts at reduction, for the reasons before given, and suggested excision of the head of the humerus. The latter procedure, he was informed, would, in all probability, accomplish the removal of the pain, but it was questionable whether he would derive any benefit from the operation as far as the use of the arm was concerned. Moreover, the operation itself involved danger, and it was to be considered whether his condition justified the adoption of a plan of treatment so formidable in character. The patient returned to his home, but in the latter part of September, 1876, again presented himself,

stating that he desired to have excision of the head of the bone performed. He felt that he could no longer endure the pain to which he was subjected, and was prepared to assume all responsibility as to the dangers of the operation. He was placed upon preparatory treatment, and instructed to return in two weeks, when the operation would be done.

In studying, in the mean time, the conditions which were present in the dislocation, and the causes concerned in the production of the pain, it occurred to me that an operation of less gravity than excision of the head of the humerus would accomplish all that was necessary. The indications were clearly to remove the pressure exerted by the head of the bone upon the vasculo-nervous cord of the axilla, portions of which had been caught between the head of the bone and the coracoid process, and were held in this position by subsequent inflammatory adhesions. Every movement of the humerus caused pain, owing to the impact of the head. It seemed to me, therefore, that relief could be afforded by destroying the continuity of the bone, so that the head would remain quiescent whilst the shaft and lower extremity could respond to the action of the muscles attached to that portion. Not only was it necessary to place the head of the bone in a state of quiescence, but it was equally, if not more, desirable to remove the constant tension produced by the contraction of the muscles acting upon the humerus. The coraco-brachialis and short head of the biceps muscles held the bone against the coracoid process, the force of contraction being exerted in the line of the long axis of the bone, whilst the muscles inserted into the bicipital groove,

assisted by drawing the shaft toward the body. I decided, therefore, to divide the bone above the insertion of these muscles, and to separate the fragments, thus destroying the continuity of the bone, so that pressure could not be exerted.

In order to accomplish section of the bone in the most exact manner and with the least risk, I determined to employ the plan of subcutaneous division, as practised by Mr. Adams, the distinguished surgeon of London, on the femur, in cases of anchylosis of the hip-joint. I was confirmed in my opinion as to the application of the operation in this case, after witnessing subcutaneous section of the femur for the relief of anchylosis, by Dr. John Ashhurst, Jr. At my request, Dr. Ashhurst saw Mr. R. with me in consultation, and agreed in regard to the employment of this method of treatment. The instruments used in effecting division of the bone were those devised by Mr. Adams, and consisted of a tenotome (Fig. 1), and a saw (Fig. 2), both with long shafts,

Fig. 1.

Fig. 2.

the cutting edge of the saw being not more than an inch and a half in extent, or equal in length to the diameter of the humerus.

The operation was performed October 16, 1876, there being present Drs. John Ashhurst, Jr., Packard, Keen, Willson, and Mr. Harrison, medical student. The patient having been placed under the influence of ether by Dr. Willson, the knife was carried flatwise through the tissues, on the outer aspect of the arm, two inches below the acromion process, directly to the bone. When the point came in contact with the bone, the position of the blade was changed, and the periosteum divided by incisions very carefully made, the edge of the knife being kept closely in contact with the bone. The knife having been withdrawn in the same manner as that in which it was introduced, the saw was passed flatwise through the opening, to the bone. The cutting edge having been placed on the bone, it was divided by very short "higgling" movements, the time required to sever it being five minutes. When section was completed, the fragments were separated, the lower one being thrown backwards and outwards, and the arm was placed in the Velpeau position and secured by bandages. Not more than a drachm of blood was lost. The wound closed in three days without suppuration. At the expiration of ten days the arm was released, and passive motion was instituted. Shortly afterwards the patient was permitted to return home, with instructions to exercise the arm daily in the various movements of abduction, adduction, rotation, elevation, flexion, etc. Immediately following the operation, the pain ceased, and he has had no return of it.

In dividing the bone I endeavored to apply the saw at the middle of the surgical neck, above the

insertion of the teres major muscle. At this point there is less danger of wounding the bloodvessels, which, above, supply the structures about the joint, and below, the humeral muscles. The anterior and posterior circumflex arteries, from the third part of the axillary, are usually above this point, and the superior profunda, from the brachial, reaches the bone at the lower border of the teres major, and then passes to its spiral groove. Application of the saw to the bone on its outer aspect, and the very restricted movements made, prevented injury to the long tendon of the biceps muscle.

Three months after the operation, the patient was present at a meeting of the College, and was examined by the Fellows in attendance. At that time the following conditions were noted: The arm hung naturally by the side; there was no apparent difference in the length of the two limbs; when stripped and examined, it was found to be natural in size and contour; the muscles were firm, and the atrophy of the deltoid had, in a great measure, disappeared. The temperature was normal; the pulse had acquired fulness, and corresponded in character with that of the opposite arm. A small red spot, two inches below the acromion process, indicated the point at which the knife had been entered. Rotation of the arm could be performed without causing pain and without moving the upper fragment. When the lower fragment was forced up and rotated, slight crepitus could be elicited. The hand could be placed on the opposite shoulder, into the pantaloons pocket, and into the hip-pocket, with ease; the watch could be taken readily from the vest-pocket; the patient used

his hand without restriction in putting on and removing his clothing, and in all of the manipulations incident to his calling as a druggist. He stated that he could raise a sixteen-pound weight by the pulley, and practised at this exercise daily, increasing gradually the weight. His general health was excellent; the depressed mental condition from which he had suffered, prior to the operation, had disappeared; and he was cheerful, and much encouraged at the prospect of being permanently relieved from pain and of having a useful arm.

A careful examination made some months later, showed that the false joint which had been formed, was in every respect satisfactory. At the time of the separation of the fragments, without doubt the periosteum was not entirely divided, but was sufficiently stripped off by the force employed, to completely prevent contact, and yet give a character of firmness to the joint, which was desirable. Moreover, the position in which the arm was placed kept the fragments at an obtuse angle, and thus assisted in the prevention of union. The movements are so free in every direction that it is quite evident that the greater portion of the circumference of the upper end of the lower fragment is free from periosteal attachments, or adventitious bands. Faithful attention to exercise of the joint, for a period covering now nine months, has placed it in such a condition that I believe no fear can be entertained as to any impairment in its functions, much less as to a total loss of movement.

In a communication which I have had the pleasure to receive from Mr. Adams, he calls attention to the fact that after section of the femur, where the opera-

tion is performed for the relief of anchylosis, "Motion is apt to be lost if passive motion is not steadily persevered in." I am inclined to believe that loss of movement would be more likely to occur in a false joint formed in the femur than in the humerus, owing to the difference in the functions of the two extremities, and to the difficulty experienced in inducing the patient to maintain steadily passive motion in the lower limb. The superincumbent weight of the body, with the contractile force of the more powerful muscular structures and a comparatively limited range of movements, would prevent the formation of a joint as freely movable as could be obtained in the bone of the upper extremity.

The results in this case have been so successful, with regard to each of the conditions to be relieved, that I have felt at liberty to report it at length, and to call attention to the subject to which it relates, which is of the highest importance, and should claim at our hands the most careful consideration.

Prof. Gross, in his System of Surgery, employs the following expressive language with regard to it:— "The subject of old, chronic, or neglected luxations, has not received the attention which its importance merits. It is to be feared that their treatment is seldom guided by sound scientific principles. They constitute a class of cases which almost every surgeon approaches with doubts and misgivings, being anxious to do something for the patient's relief, and yet afraid lest that something shall produce serious, if not irreparable mischief." These feelings he himself has shared, and he further declares the dangers which attend the usual methods of treatment to be

such as to cause the surgeon "to hesitate before he enters upon an enterprise so fraught with unpleasant consequences." Under the head of Chronic Dislocations of the Shoulder-joint, he calls attention to the fact of their being frequently brought under the notice of the surgeon, and of the necessity of using great caution in undertaking efforts at reduction. Whilst the records of surgery contain numerous instances of reduction in cases of long standing, they present on the other hand a long list of disasters which have followed such attempts. It is fair to assume that this list is not complete, owing to the lack of courage on the part of many operators to report accidents of such a grave nature. On examination it is found that the reported cases have occurred in the hands of some of the most eminent men in the profession, men whose standing could well endure the shock of such misadventures; what is the record among the mass of practitioners under whose treatment most of the cases may have passed?

The nature of the injury inflicted by efforts at reduction has varied. In the greater number of cases, rupture of the artery or vein, or both, has occurred; in some, the nerves have been lacerated; in others, fracture of the bone has resulted, with more or less injury to the surrounding structures. In the notable case reported by Guérin, the entire forearm was torn from the body in the efforts to reduce a luxation of only three months' standing. In many cases in which the attempts have been successful in restoring the head of the bone to its normal position, the force employed has been so severe as to permanently impair the usefulness of the joint.

Returning to the consideration of those cases of long standing in which reduction has been successfully accomplished, I think we are justified in subjecting them to critical examination before accepting the results. It is important, first, to have determined the exact nature of the dislocation; Was it complete in character, by which I mean, did there exist a rupture of the ligaments to such an extent as to permit the escape of the head of the bone from its enveloping ligament, and its permanent lodgment in an abnormal position? Second, what was the condition of the joint after restoration of the bone to its place? The case reported by Dr. Nathan Smith, in which reduction was effected after luxation had existed for one year, is quoted as a remarkable instance in all text-books on surgery. Desiring further information with regard to this case, I addressed a communication to Dr. Smith's son, the late venerable Dr. N. R. Smith, the distinguished surgeon of Baltimore, and was informed by him in reply that, as far as he remembered, the case occurred in the person of a woman of delicate constitution, and in whom the muscular tissues were much relaxed. Within a few months past, I was asked by my friend Prof. Tyson, to examine a chronic luxation of the shoulder-joint of over two years' duration, in a woman upwards of sixty years of age. In this case, I found the tissues so relaxed that it was possible not only easily to reduce the dislocation, but to produce one at will. If the cases of chronic character, reported in the treatises on surgery as instances of successful reduction, partook of the nature of these above alluded to, then

they must be received with caution, "especially when viewed as examples for our imitation."

With regard to the condition of the articulating surfaces of the bones entering into the formation of the joint, I cannot conceive it possible that they should remain in a state so normal as respects function, as to admit of the readjustment of the parts, and a return to usefulness, after so long a lapse of time. How much in accord with this fact is the work which nature herself accomplishes in these very cases, as observed in the cavities formed for the reception and lodgment of the displaced bone in the various parts of the body adjacent to the articulation? By her laws, a part of the body not performing its accustomed function, is not maintained; as witness the atrophy occurring in the muscular structures in palsied extremities, or the molecular destruction of detached and dead osseous tissue. Does it not so follow with the unused joint? Does not the function of synovial membrane cease, and the membrane itself disappear; the ligaments become rigid; the muscles atrophied? Is it, therefore, under any circumstances, a rational plan of treatment to restore the displaced bone to a cavity the character of which must have become completely changed?

In order to facilitate reduction in chronic luxations, Diffenbach suggested, and, it is stated, performed with success, subcutaneous division of the muscles, ligaments, and tendons surrounding the joint; the case was one of two years' standing. This operation I have witnessed once, and it was followed by a fatal result. Certainly it is a procedure which cannot be commended; and it is interesting to contemplate the

condition and capacity for use of an articulation which has been submitted to such general mutilation of its component parts. As another method of treatment, and one more rational, excision of the head of humerus has been performed with success. Two such operations have been done in this country, and in each case the operation was called for to afford relief from constant pain produced by pressure. The operation, it is to be remembered, involves much danger, the state of the parts differing materially from that encountered where excision is called for in cases of wound of the articulation.

Such, therefore, being the record under the plans of treatment usually suggested and executed in cases of chronic luxation of the shoulder-joint, I feel that subcutaneous section of the bone can be offered as a method which, in its simplicity, freedom from hazard, and successful result, as exemplified in the case reported, commends itself. When operative interference is demanded for the relief of pain consequent upon pressure, subcutaneous section accomplishes the result by destroying the continuity of the bone. In cases where the impairment of the functions of the arm is so great as to justify efforts to secure improvement, section of the bone gives relief in the formation of a new articulation, the use of which approaches in character that of the normal joint.

Further experience will test the claims of the operation, and develop its defects, if they should exist.[1]

[1] It should have been stated that Prof. Agnew also saw this patient in December, 1875, and confirmed the diagnosis, and agreed in advising that no effort at reduction should be made.

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